UNIVERSITY OF MICHIGAN
AUG 22 1952
MEDICAL LIBRARY
DOES NOT CIRCULATE

BRITISH MEDICAL JOURNAL

JOURNAL OF THE BRITISH MEDICAL ASSOCIATION

SATURDAY AUGUST 9 1952

TABLE OF CONTENTS

Surgery of Peptic Ulceration SIR HENEAGE OGILVIE, K.B.E., D.M., M.Ch., F.R.C.S. 299

Treatment of Massive Bleeding from Peptic Ulcer
A. G. OGILVIE, M.D., F.R.C.P., N. CARDOE, M.B., B.S., and F. H. BENTLEY, M.D., F.R.C.S. 304

Malaria Eradication Scheme in Mauritius M. A. C. DOWLING, M.R.C.S., L.R.C.P. 309

Superiority of "Camoquin" Over Other Antimalarials
INDER SINGH, M.B., F.R.F.P.S., and T. S. KALYANUM, M.B., B.S. 312

Observations on Methods of Measuring the Erythrocyte Sedimentation Rate
A. GOLDBERG, M.B., F.R.F.P.S., M.R.C.P., and HUGH CONWAY, M.B., M.R.C.P. 315

Salm. typhi Osteomyelitis JOHN FERRIER, M.B., F.R.F.P.S. 318

Paratyphoid B Osteomyelitis of Frontal Bone E. PEYSER, M.D., and M. MADORSKY, M.D. 320

Apparatus for Demonstrating Hazards of Static Electricity in Operating Theatres
K. C. MAGEE, M.I.Mar.E., and A. BRACKEN, Ph.D. 321

MEDICAL MEMORANDA

Transmesenteric Herniation. JOHN A. BATY, F.R.C.S.Ed. 322
Transmesenteric Herniation due to Tuberculous Mesenteric Adenitis. J. A. RUSSELL JOHNSON, M.B., F.R.C.S. 323
Spontaneous Rupture of the Stomach. R. REJTHAR, M.D. 324

REFRESHER COURSE

Anaesthetic Explosions. C. F. HADFIELD, M.D., D.A., F.F.A. R.C.S. 332

GENERAL ARTICLES AND NEWS

CHIEF CHARACTERISTICS OF NERVE GAS ..334
MEDICAL NOTES IN PARLIAMENT 344
MEDICO-LEGAL 344
UNIVERSITIES AND COLLEGES 345
MEDICAL NEWS 347
BIRTHS, MARRIAGES, AND DEATHS 348

REPORTS OF SOCIETIES

ROYAL SOCIETY OF TROPICAL MEDICINE AND HYGIENE: A New Antimalarial ..335

REVIEWS

Atlas of Bone Disorders 325
Bone and Joint Diseases 325
Management of the Newborn 325
Body Movement 326
U.S. Public Health Service 326
Medical Symptoms 326

LEADING ARTICLES

Criticism of Working Party's Scheme ..327
Closed Book 328
Problem of the Feeble-minded 329

ANNOTATIONS

Treatment of Leprosy 330
Progress in Malaria Control 330
Radiography of the Lungs 331

QUESTIONS AND COMMENTS

Admission of Poliomyelitis Cases to General Wards—Sun-glasses—Aortic Incompetence in Marathon Runner—Acetylcholine in Anxiety States—Atheroma and Cerebral Accidents—Cervical Spondylo-arthrosis — Head Injury and Drunkenness—Snags in Bacteriological Diagnosis of Tuberculosis—Tinnitus in the Elderly—Congenital Cataract—Brain Foods—Coal Tar and Pine Tar Soap—Drunk in Charge 349-50

SUPPLEMENT

Full Contents on First Page of Supplement

The Danckwerts Award 91
Durham Closed Shop 91
Health Service in Eire 92
N.O.T.B. Association 92

CORRESPONDENCE

Trials of Antihistaminics. W. A. BAIN, Ph.D.; O. A. N. HUSAIN, M.D. 337
Future of Public Health. W. S. CRAIG, M.D. 338
Training for District Nurses. O. LL. LANDER, M.B. 338
W.H.O. and Birth Control. F. H. STEVENSON, M.D.; D. A. T. TIZARD, B.M.; ANDREW MALLESON 338
Malaria in Britain. J. E. JELINEK, M.B. 339
Malaria Control in Mauritius. M. A. C. DOWLING, M.R.C.S. 339
Pain in Childbirth. F. NEON REYNOLDS, F.R.C.S.Ed. 339
Gastric Gossip. A. J. GLAZEBROOK, M.D. 339
Peptic Ulcer in the East. M. L. H. FLINDT, M.B. 339
Sympathectomy for Raynaud's Disease. H. A. HAXTON, Ch.M. 340
Treatment of Intersex. D. INNES WILLIAMS, F.R.C.S. 340

OBITUARY

G. Carmichael Low, M.D., F.R.C.P. 341
L. Cunningham, M.B., F.R.C.P. 342
Sir Hugh Cairns, K.B.E., D.M., F.R.C.S. ..342
Catherine Chisholm, C.B.E., M.D., F.R.C.P. 342
George Mitchell, M.B. 343
S. Y. Baird, M.B. 343

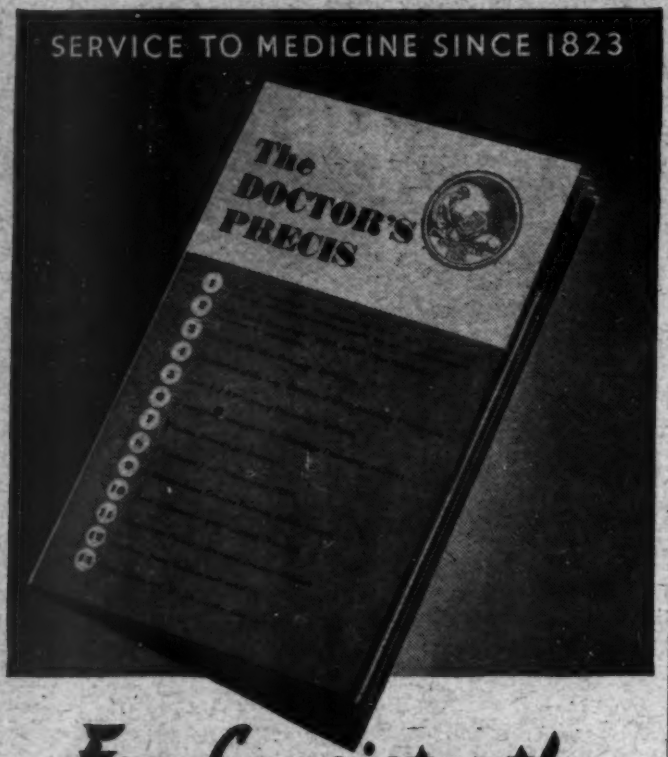
Vital Statistics 346

BRITISH MEDICAL ASSOCIATION, TAVISTOCK SQUARE, LONDON, W.C.1

No. 4779, WEEKLY, PRICE 2/-

Copyright

REGISTERED AS A NEWSPAPER



For Consistently High Quality

Over 125 years ago Dalmas began serving the needs of the medical profession for adhesive dressings and strappings. The "Doctors Precis" Book (illustrated) gives full information on Dalmas products — may we send you a copy? The following products manufactured by Dalmas are available on E.C.10's.

DALMAS
MEDICAL & SURGICAL PRODUCTS

- Dalzo Zinc Oxide Plaster, B.P.C. on cloth.
- Dalzo Zinc Oxide Felt, Self Adhesive.
- Dalzoflex (Regd.) Elastic Adhesive Bandages.
- Lestreflex (Regd.) Elastic Diachylon Bandages.
- Dalzo-Lastic Wound Dressings. (D.T. Specification.)
- Dalzo-Lastic Boil Plasters.
- Dalmas Belladonnæ Porous Plasters.
- Dalzoband Zinc Paste Bandage and with Ichthyol.
- National Corn Plaster and Crescent Corn & Bunion Rings.
- Permalast Cotton Elastic Bandages, N.H.S.
- Petronet Petroleum Jelly Gauze Dressings.
- Sulphonet Sulphathiazole 5% Paraffin Gauze Dressings.
- Nu-san Jelly Antiseptic.
- "Sona" Asthma Tablets & Inhalant.

A Copy of the DALMAS "DOCTOR'S PRECIS" BOOK will be supplied on request.

MADE BY DALMAS LTD., LEICESTER & LONDON, ESTD. 1823

Tried and True-
★ Made from the finest Sheffield steel, Swann-Morton surgical blades are individually tested for keenness and flawlessness —then sterilised and coated with pure Vaseline to reach the surgeon's hands in perfect condition. Handles are of stainless metal, precisely machined to ensure that blades fit accurately and rigidly. There are eleven types of blade, as illustrated, and three types of handle.
Swann-Morton
W. R. SWANN & CO. LTD · PENN WORKS · SHEFFIELD · ENGLAND

FINANCE
for the acquisition by
PAYMENTS OUT-OF-INCOME
of
SURGERY AND OTHER FURNITURE, SURGICAL INSTRUMENTS, MEDICAL TEXT BOOKS, X-RAY APPARATUS, MOTOR CARS

The above list is illustrative only. Under its equipment Purchase Plan, the company is prepared to assist doctors to acquire ANY article and spread the cost over a period.

BRITISH MEDICAL FINANCE LTD.
Tavistock House South, Tavistock Square, London, W.C.1

ERRATA

SULPENIN

Owing to a printer's error in the advertisement for Sulpenin in the *B.M.J.* dated July 26 the formula appeared as

Crystalline Penicillin G (Potassium Salt) 1,000 units
Sulphamerazine 0.25 gramme
Sulphadiazine 0.25 gramme

The correct formula is

CRYSTALLINE PENICILLIN G (POTASSIUM SALT) 100,000 units
SULPHAMERAZINE 0.25 gramme
SULPHADIAZINE 0.25 gramme

Any confusion this error may have caused is regretted.

ALLEN & HANBURYS LTD.

BRITISH MEDICAL JOURNAL

LONDON SATURDAY AUGUST 9 1952

THE SURGERY OF PEPTIC ULCERATION*

BY

Sir HENEAGE OGILVIE, K.B.E., D.M., M.Ch., F.R.C.S.

Surgeon, Guy's Hospital and Royal Masonic Hospital

All the best people have peptic ulcers. According to estimates based on post-mortem statistics and on industrial surveys, one in every ten adults in Britain is so afflicted. These sufferers are, on the whole, the ablest, the most hard-working, and the most conscientious members of the community. Their lives are made miserable and their working efficiency is diminished by pain and indigestion, and they come to us for relief.

Peptic ulceration is disabling but not necessarily fatal, and it is therefore our duty to advise that treatment which is most safe, simple, and satisfactory. Medical treatment, which has, or rather which admits of, no mortality, is therefore the first choice. It is still the first choice, but since the end of the war there has been a notable swing of opinion towards surgery, a tendency to resort to it more often and earlier. The reason for this change is twofold—that medical treatment, though simple and safe, has not proved particularly satisfactory, and that surgical treatment, though it is less simple than formerly, is very much safer and more satisfactory than it used to be.

Published statistics are the basis of all unsound teaching, for they never present average results, but only those so much better than the average that they induce the doctor to collect them and the editor to give them space. Those I give you are from a good non-teaching hospital; they were prepared six years ago by one of my registrars, who is now a professor of surgery, and they are truthful.

1,000 Consecutive Admissions for Peptic Ulceration

For perforation	60 cases
For haemorrhage	215 ,,
For jejunal ulcer	65 ,,
For uncomplicated peptic ulcer	660 ,,

Analysis of the 660 Uncomplicated Cases

Treated medically	400 cases
Died under treatment	1%
Satisfactory after 5 to 10 years	34%
Unsatisfactory after 5 to 10 years	56%
Died between discharge and 10th year	10%
Treated surgically	260 cases
Died under treatment	7%
Satisfactory after 5 to 10 years	70%
Unsatisfactory after 5 to 10 years	20%
Died between discharge and 10th year	10%

These results are poorer than the average, for the hospital concerned is one that admits a large number of old and infirm patients, and the surgical treatment was not in accord with modern ideas—for instance, 48% of the operations performed were gastro-jejunostomies; nevertheless they emphasize the difference between medical and surgical treatment. Medical treatment has a low primary mortality, but the late results are unsatisfactory; surgical treatment has a higher hospital mortality, but of those who survive the majority are relieved of their symptoms.

Rational treatment of any disease depends on a knowledge of its cause. We know nothing of the cause of peptic ulceration. We know, it is true, that peptic ulcers are found only on surfaces exposed to the action of gastric juice containing free hydrochloric acid, that in ordinary circumstances they are limited to the district between the cardia and the bile papilla, but that they may be found in any organ or structure which is brought into contact with gastric juice by sphincteric deficiency, ectopic secretion, or surgical anastomosis—in the oesophagus, in Meckel's diverticulum, or in the jejunum, the colon, the gall-bladder, or the skin after short-circuit operations or gastrostomy. But we know also that acid is part of the normal mechanism of gastric digestion, and that it does not attack the walls of its compartment unless it is secreted in amounts and at times that have no relation to the needs of digestion or unless those walls are already damaged. Acid is the cause of persistence rather than the cause of origin of peptic ulcer.

Gastric and Duodenal Ulcers

We are agreed that the treatment of peptic ulceration is medical in the first place. In discussing the surgery of peptic ulcer, therefore, we must attempt to define at what point the physician retires and the surgeon steps in. We cannot do this without first breaking down the peptic ulcer group into the gastric ulcers and the duodenal ulcers. These two lesions have certain features in common: they are found in the acid-containing portions of the alimentary tract, and their leading symptom is pain related to the taking of a meal (Fig. 1). In all other respects they are different. Gastric ulcer is rarer than duodenal ulcer but equally common in both sexes; duodenal ulcer preponderates in males. Gastric ulcer starts at from 35 to 40 years of age, duodenal ulcer some ten years earlier. Gastric ulcer is usually associated with a long stomach of poor tone emptying slowly and with a level of acid secretion below the average, duodenal ulcer with a high stomach emptying rapidly and a high acid curve.

Gastric ulcer seems to be commoner in the underprivileged and underfed, though the wealthy gourmet is not immune, but its cause is quite unknown; duodenal ulcer is definitely associated with a certain physical and psychological type, the lean, wiry, ambitious, worrying, conscientious, heavy-smoking intellectual, and the

*A B.M.A. Lecture given at Pontypridd on February 28.

first onset and subsequent relapses are usually associated with periods of stress, with anxiety, frustration, and over-smoking. A gastric ulcer yields well to medical treatment when it is small and shallow, but after several relapses it becomes fibrous, adheres to surrounding structures, and deforms the stomach wall, so that it tends to relapse as soon as normal diet is resumed. A duodenal ulcer reacts equally well to treatment in the early stages, and will remain healed if the stress and the smoking can be permanently eliminated; in healing it may leave a scar, but a scarred duodenum can carry out its functions with complete satisfaction to its owner so long as the scar has not narrowed the lumen enough to obstruct the onward passage of a meal. Gastric ulcers may become malignant, duodenal ulcers never.

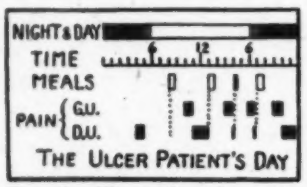


FIG. 1.—The pain of peptic ulcer.

To sum up: in gastric ulcer the cause of origin is unknown, but the cause of persistence is in the stomach itself; in duodenal ulcer, the cause of origin and of persistence are in the patient, in his psychological make-up, his job, and his habits, and in the reaction of each upon the others. Therefore in gastric ulcer medical treatment should be abandoned as soon as it is clear that it has not produced permanent healing; in duodenal ulcer medical treatment should be persisted in until it is clear that it cannot succeed. Put in another way, gastric ulcers should be taken from the physician after twelve months if he has failed to heal them, or earlier if there is any reason to doubt their innocence; duodenal ulcers should be kept away from the surgeon till the patient is 45, or till some complication has arisen that can be treated only by operation.

Malignancy

We operate on gastric ulcers early and with enthusiasm because the prospect without operation is one of increasing misery. The stomach is a reservoir that expands to contain whatever the oesophagus brings it and that sinks lower in the abdomen as it fills; it is also a muscular mill that churns up the food and mixes it with the digestive juices. When an ulcer has produced sufficient scarring to prevent free movement and smooth peristalsis, the stomach can function painlessly only when its owner is in bed and taking frequent small meals that do not need churning—that is, during periods of medical treatment; as soon as an ambulant life and normal meals are resumed the ulcer is dragged upon and becomes active and pain returns. A gastric ulcer is under suspicion of malignancy if it is an inch or more in diameter and does not rapidly shrink under medical treatment, if it is in the prepyloric inch or on the anterior wall or the greater curve, or if it is accompanied by loss of appetite. Whether a true peptic ulcer ever becomes malignant is open to question, and the estimates of the frequency of such a change vary from the 50% of some American writers to Newcomb's 3%.

I myself have never seen an undoubtedly innocent peptic ulcer change to a cancer, but in about 10% of my gastrectomies for gastric ulcer I am told by the pathologist that the specimen shows an ulcer-cancer, meaning an ulcer with cancer cells on one edge and the typical histological appearances of a chronic peptic ulcer at the other. I am, however, unable to accept spatial propinquity as proof of causal relationship, and if there is a relationship I believe it is the cancer that caused the ulcer rather than the ulcer that caused the cancer. When we go back from the pathologist's report to study the clinical history of the case we find that it is never typical of an innocent peptic ulcer. The age of onset has been unusually late, the pain has been more constant and more irregular in its relation to meals, the response to treatment has been unsatisfactory, and the remissions have been brief. I believe that all, or the great majority, of these ulcer-cancers are scirrhous cancers which have been attacked by the gastric juice and have undergone the usual process of healing in those parts where all malignant cells have been digested away, just as the scirrhous cancer of the breast may heal over after it has been burnt by the caustics of the cancer quack.

For our present purpose it is sufficient to emphasize that the distinction between an innocent and a malignant gastric ulcer cannot be made on clinical or radiological evidence, and that many of those that appear for the first time after the age of 40 are really wolves in sheep's clothing. The only safe course is to advise gastrectomy in all gastric ulcers that are not rapidly and permanently healed by rest and diet. We can do this without hesitation because we know that the patient will live happily ever afterwards. The failures of gastric surgery are all after operations for duodenal ulcer.

Duodenal Ulcer

In duodenal ulcer we are dealing with a lesion that is not necessarily progressive. The duodenum seems to be a passage-way and a mixer of enzymic cocktails for the second phase of digestion which is to follow, rather than part of the digestive laboratory, and it can carry on this function even though it be scarred, provided the ulcer is not active.

In discussing the cause of duodenal ulceration I speak only of the ulcers that I know. The duodenal ulcers of Madras—extensive lesions besides which the ulcers of the London stockbroker seem trivial—occur in peasants who do not smoke and do not worry, and they may be due to the liberal use of red pepper. The duodenal ulcers of the United States and of my native land, to judge from the writings of American and Scottish surgeons, differ in some respects from those of England and Wales, which seem to be manifestations of the stress syndrome or, to use a more modern term, of the adaptation syndrome. Duodenal ulcers as we see them occur in those who are trying to "fill the unforgiving minute with 60 seconds' worth of distance run"; they start at peak periods of stress, they tend to become quiescent when stress is relieved and to flare up again when it is renewed, and they can heal and remain healed permanently if the stress is removed by domestic adjustment, by worldly success, or by the philosophy that comes with advancing years.

If a duodenal ulcer is, as we believe, a local manifestation of a general disease—the symptom of a turmoil that affects alike the cerebrum, the mid-brain, the autonomic system, and the hormones which they control—the treatment is primarily that of the patient and only incidentally that of the lesion. The physician, the psychiatrist, the wife, the business associate, the trusted friend, all have their part to play, and the surgeon should be right out of sight in the wings awaiting his cue to come on. The cue comes when certain complications arise—perforation, stenosis, or major haemorrhage—or when it is clear that medical treatment is failing or is bound to fail. The difficulty lies in determining when, in the absence of danger signals, the orange light that precedes the red has begun to show.

Indications for Operation

Perforation cannot be anticipated; in many cases the ulcers that perforate are acute ones with no previous history. Stenosis can, however, be anticipated, and if in an old-standing ulcer there is radiological proof of delay in emptying or of failure in gastric tone at a time when the ulcer is clinically quiescent, it is wiser to anticipate the inevitable by operating before the patient has started to lose weight and strength. A single major haemorrhage—that is, one in which two or more pints of blood have been lost within an hour or so—is an absolute indication for operation in a man of 50 or over with a known duodenal ulcer; but lesser

haemorrhages in younger men, if they have been repeated more than twice within the space of two years, are a call for surgery rather than optimism. The greatest difficulty lies in giving exact definition to that commonly cited indication, "the failure of medical treatment," for it may not be the treatment that has failed, but the patient that has failed the treatment. Those poor fish who have not the moral fibre to obey instructions to re-order their lives or to curtail their indulgences are the physician's headache; handed over to the surgeon they provide the majority of the dumping syndromes.

I work on three criteria. The first of these is age. When a man has reached 50 and must still govern his life and restrict his pleasures in order to keep his ulcer quiet the time has come for surgery. He has some 20 years to live, and they are perhaps the most important ones, for the goal towards which he has been striving is now within reach and the leisure for which he has worked so long is his to enjoy. After gastrectomy he will enjoy it. Once again he can work and sleep without fear of pain. Once again he can run his eye down the menu and select whatever takes his fancy. So uniformly good are the results of gastrectomy that I have recently dropped the minimum age for elective operation from 50 to 45, and I shall probably bring it down to 40. My second criterion is loss of earnings, and my third is loss of weight. If a man who is following instructions conscientiously has nevertheless lost 10% of working time or 10% of weight within the previous 12 months I should advise operation.

Aims of Surgery

The aims of surgery, in the most general terms, are to relieve the patient permanently of the symptoms of which he is complaining. The patient suffering from peptic ulceration complains of pain and indigestion; to relieve these permanently we must do three things: first, we must remove the ulcer or at any rate remove it from the acid pathway; secondly, we must overcome the effects the ulcer has produced, chiefly those due to scar tissue; and, thirdly, we must remove the cause of ulceration or at any rate remove the likelihood of further ulceration. We do not know the cause of peptic ulceration, but we do know that chronic ulcers occur only in the presence of free hydrochloric acid. Any operation for the treatment of ulcer must therefore have as one of its objects the permanent reduction of acid secretion to a level at which fresh ulceration is unlikely.

Papers on the causes of peptic ulceration, for the most part based on experiments on animals whose dietetic habits have no resemblance to those of man, are by now innumerable. Each year a fresh crop descends on us like leaves in autumn. As a group they are self-cancelling, for it is possible by selection among them to support any pathological theory, however improbable, and any operation, however bizarre. Very few surgeons can resist for long the temptation to tell their fellows how to repair the inguinal canal and how to cure peptic ulcers. Among the operations that have been described, many of which betray the influence of Heath Robinson rather than that of John Hunter, we need discuss three groups only, all of which have as their purpose the reduction of acid: short-circuiting operations; vagotomy, alone or combined with other procedures; and gastrectomy.

Short-circuiting Operations

The short-circuiting operations—the various gastro-jejunostomies, pyloroplasties, and gastro-duodenostomies—attempt to neutralize the gastric acid by the alkaline contents of the duodenum, and they were first proposed in the belief that such neutralization is part of the normal mechanism of digestion. We now know that in man digestion is carried out in four distinct departments, separated by the pharyngo-oesophageal sphincter, the pyloric sphincter, and the ileo-caecal sphincter, and that normally nothing which has once passed any sphincter is allowed to return. Gastric digestion is brought to a close, when the meal is in a state to pass onwards, by the shutting down of acid secretion and the neutralization of any free acid that remains by the alkaline gastric secretion, and reflux from the duodenum occurs in appreciable amounts only when there is some distal obstruction. The short-circuiting operations are therefore wrong in theory, and in practice it is found that many are failures from the start and few give relief for more than four to five years.

The passing of gastro-jejunostomy marks the end of an epoch. No operation has in its time done more for surgery. No other operation has been so polished and perfected by generations of surgeons that it has become a standardized technical masterpiece, performed in much the same manner by surgeons in all parts of the world. But it stands condemned, both on theory and on its own results. Gastro-jejunostomy was claimed to reduce acid "by Nature's method," the pouring of duodenal contents into the stomach, and it was shown to do so by post-operative test meals. We now know that duodenal regurgitation is not a natural process, and we know that the drop in acid which follows a short-circuit is merely one indication of the general inhibition of gastric function that any recent trauma will produce, and that a test meal done a year later will show an acid secretion back to its pre-operative level or higher. Gastro-jejunostomy will heal a duodenal ulcer by diverting the acid chyme from the ulcer site, but in doing so it transfers the factors that caused that ulcer to another site in an intensified form. In normal digestion acid is secreted only while food is in the stomach, first by vagal secretion, and later, when appetite is sated, by the action of a hormone secreted in the pyloric antrum as the products of digestion reach it (Fig. 2).

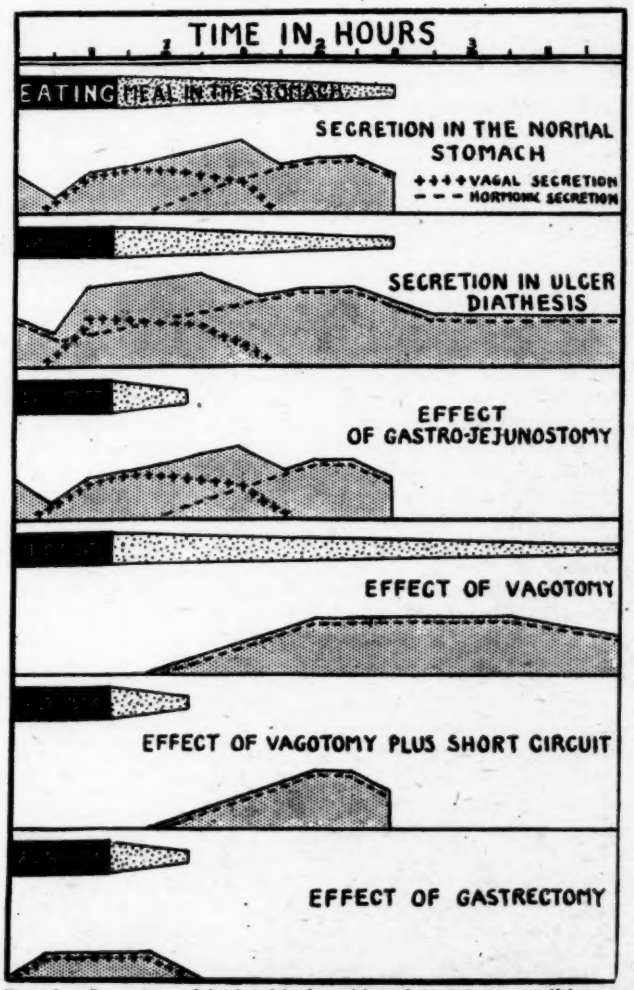

TIME IN HOURS
EATING MEAL IN THE STOMACH
SECRETION IN THE NORMAL STOMACH
++++ VAGAL SECRETION
- - - HORMONIC SECRETION
SECRETION IN ULCER DIATHESIS
EFFECT OF GASTRO-JEJUNOSTOMY
EFFECT OF VAGOTOMY
EFFECT OF VAGOTOMY PLUS SHORT CIRCUIT
EFFECT OF GASTRECTOMY

FIG. 2.—Secretion of hydrochloric acid under various conditions. (This diagram makes no claim to accuracy or to indicate definite values, but is merely meant to illustrate the views expressed in the text.)

After gastro-jejunostomy the food leaves the stomach in 20 minutes to half an hour, and for the next hour the second hormonic tide of unwanted and harmful acid is poured into an empty stomach and out over an empty stoma. In practice stomal ulceration of some degree is found in at least 20% of gastro-jejunostomies done for duodenal ulcer and observed for more than three years, and the number thereafter increases yearly, so that in post-mortem series over half the patients with short-circuits can be shown to have had ulceration at the stoma. It used to be thought that gastro-jejunostomy was a safe operation in pyloric stenosis with hypochlorhydria or achlorhydria; unfortunately this is not so. The reduction in acid is evidence of gastritis, and the gastritis usually recovers rapidly once the stasis that caused it is overcome. Gastro-jejunostomy still has a place as a palliative operation in cancer of the stomach, and as a simple expedient to overcome stenosis in those so old and feeble that they cannot be expected to survive for three years. As a leading operation for the treatment of peptic ulceration it must go, displaced but not discredited, as is the unhappy fate of discarded political leaders.

> But yesterday the word of Caesar might
> Have stood against the world; now lies he there,
> And none so poor to do him reverence.

Vagotomy

Several surgeons have divided the vagi for gastric pain. Lester Dragstedt deserves the credit for some outstanding experimental work on the effects of vagal section in animals. He isolated the stomach with its vascular and nervous supply intact, restoring the food channel by anastomosing the oesophagus to the duodenum, and demonstrated that in the stomach so isolated division of the vagi reduced the quantity and acid level of the gastric secretion by about one-half. He advocated vagotomy as a reasoned and safe procedure for the treatment of peptic ulceration. In the safety of vagotomy lies its chief danger, for it is eagerly practised by surgeons who should not be allowed to operate at all. I believe vagotomy to be dead but not yet safely buried. Fifteen years ago it shot up like a rocket into the gastro-enterological sky; it blazed into a cluster of stars and we all said, "Ooh!"; then the stars died and the stick came down. It only remains to burn the stick.

It is possibly significant that of Dragstedt's recent publications as many are in defence of vagotomy against its attackers as in praise of its merits. It is equally significant that the surgeons of the Mayo Clinic, who have never been known to say an unkind word against anybody or anything, are receiving the failures of vagotomy in increasing numbers, and have practically dropped it from their repertory. In the last annual report of the Mayo Clinic, under "Surgery of the Stomach and Duodenum," the following comments are made about vagotomy. "The relative percentage of vagotomy with and without associated gastric operations to the percentage of various operations used in the surgical treatment of duodenal ulcer has decreased steadily since 1947 (6% in 1950 as compared with 15% in 1947). Only one factor can be accepted as an explanation for this sharp decrease, and that is the gradually increasing dissatisfaction with this procedure in the primary surgical treatment of a patient who has a duodenal ulcer except in relatively unusual circumstances."

Vagotomy is unsound, in the way that any procedure based chiefly on animal experiments is apt to be unsound (Fig. 3). It is unsound in that it cuts out the nervous secretion of acid, which is harmless because it is produced in response to the sight and taste of food and is therefore buffered by food, while it leaves untouched the hormonal secretion. It is still unknown whether the unwanted secretion of acid which is so characteristic of ulcer diathesis is vagal or hormonic in origin, but most stress diseases are characterized by hormone overaction. Vagal section cuts out early acid secretion, but it also cuts out gastric motility, and in most cases some form of short-circuit is necessary to overcome the resultant stasis. When the stasis is thus relieved by gravity emptying, the stomach and the stoma, unprotected by food, are at the mercy of the hormone-induced acid secretion. The hormone secretion can be abolished by pylorectomy, but the patient is then reduced to a state of complete and permanent achlorhydria, with all its attendant risks of repeated infection and of cancer. In my view gastro-jejunostomy still has a small but recognized place in the surgical programme, but vagotomy has none.

Gastrectomy

Gastrectomy is at present the operation of choice for the treatment of peptic ulceration, whether gastric or duodenal. It is based on sounder reasoning and can show far better results than any alternative procedure, and it will therefore remain until something that can be shown to be better comes to replace it. Gastrectomy, properly planned and executed, lowers the acid secretion to a safe level without abolishing it and restricts that secretion to the time that the meal is in the stomach; it thus retains gastric secretion but abolishes the risk of re-ulceration. A gastrectomy for the treatment of peptic ulceration must satisfy five criteria:

1. The whole of the pyloric mucous membrane must be removed to abolish hormonic secretion, the chief factor in primary and recurrent ulceration.

2. The body of the stomach, carrying the highly convoluted areas of acid-forming mucous membrane, must be removed, but enough stomach should be left to make a cavity that will contain a pint, a test it will often be called upon to perform when the enjoyment of life returns.

3. The perigastric tissues on the lesser curve, but not necessarily the muscle coats, must be divided at the level of the cardia in order to divide the leash of vagal branches that spray along the lesser curve, and in order to release the stomach from the tethering of the left gastric artery and to give to the reduced cavity that free motility which all functioning organs should possess.

4. The stoma should not exceed the size of a two-shilling piece, that of the normal jejunum when fully distended.

5. The stoma should be so arranged that the meal, on leaving the stomach, can pass in an onward direction only.

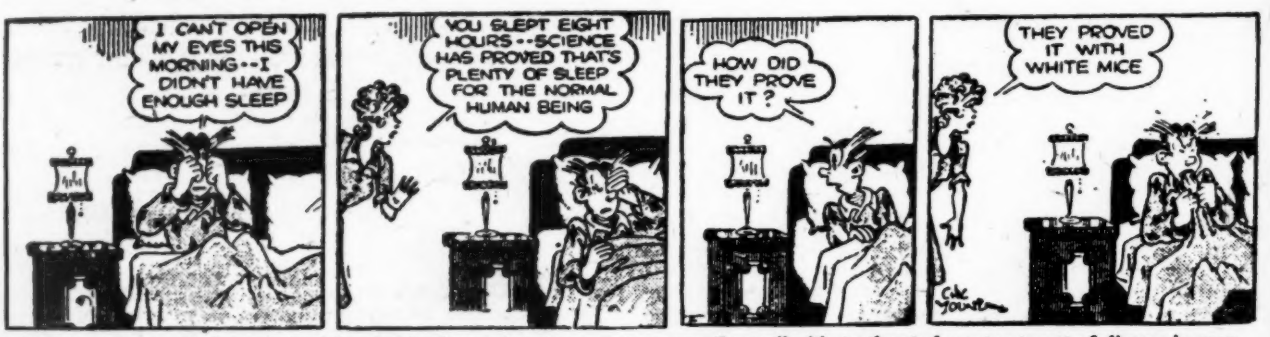

I CAN'T OPEN MY EYES THIS MORNING--I DIDN'T HAVE ENOUGH SLEEP
YOU SLEPT EIGHT HOURS--SCIENCE HAS PROVED THAT'S PLENTY OF SLEEP FOR THE NORMAL HUMAN BEING
HOW DID THEY PROVE IT?
THEY PROVED IT WITH WHITE MICE

FIG. 3.—The conclusions derived from animal experiments are not necessarily applicable to the study or treatment of disease in man. (By courtesy of King Features Syndicate and the *Daily Graphic*.)

These requirements can be met in gastrectomy of two types—in the Billroth I, in which the reduced stomach is anastomosed to the duodenum, and in the Billroth II, in which the stomach is joined to the jejunum, with a Lake–Finsterer valve to prevent regurgitation and a reduced stoma to prevent dumping.

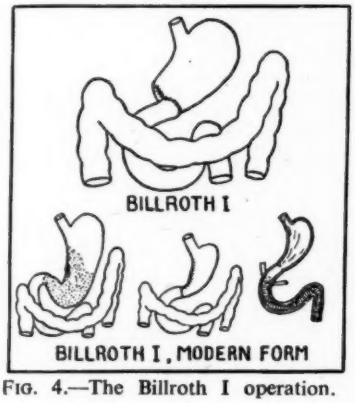


FIG. 4.—The Billroth I operation.

The Billroth I (Fig. 4) is an excellent operation. Its advantages are that the infracolic compartment of the abdomen is not interfered with in any way, that the food on leaving the stomach can go no other way but onwards (Fig. 5), and that the gastric contents are poured into a part of the intestine protected by Brunner's glands against acid damage. The operation, however, is uniformly successful only when the duodenum is normal—that is, when the ulcer is in the stomach. So good are the results in gastric ulcer that surgeons are sometimes tempted to use it in the treatment of duodenal ulcer. A skilful technician can manage to dissect a scarred duodenum right down to the bile papilla and afterwards anastomose the stomach to the duodenal stump, and he takes pride in doing so. But the duodenum he uses has few Brunner's glands left, and the stoma he fashions, joined to a short duodenum and laid in the bed of an ulcer or the raw areas of a dissected pancreas, has none of the mobility which a happy stoma should possess. There are some good results, but there are too many bad ones to justify the continued use of this operation for duodenal ulcer when the Billroth II (Figs. 6 and 7) is so satisfactory.

Such difficulties as there are in the performance of the Billroth II gastrectomy are entirely concerned with the closure of the duodenal stump. In most cases this is easy, in some it is difficult, in a few it is impossible; and he is a wise surgeon who recognizes the impossible task before it is too late. Two devices are recommended to circumvent the difficulties of the fixed oedematous and adherent duodenum. The first is Bancroft's operation—division of the muscular coats of the pyloric antrum, dissection of the mucous coat down to the pyloric ring, division and suture of the mucous membrane at this level, and closure of the sero-muscular coats over the stump. This device has the disadvantage that the ligation of the vessels in the submucous layer damages to some extent the blood supply of the outer coats, and healing is not always uneventful. In London, at any rate, Bancroft's operation is in disfavour. The second alternative is to divide and close the pyloric antrum 2½ in. (6.3 cm.) proximal to the pylorus and then perform a standard high gastrectomy; six to eight weeks later the abdomen is reopened, when the oedema will be found to have disappeared, the adhesions to have become avascular, and the dissection and closure of the duodenum to be a simple matter (Fig. 8). The second operation is essential, for, while the pyloric mucous membrane remains, recurrence at the stoma is always possible.

FIG. 5.—The Billroth I operation; post-operative skiagram.

I have spoken of gastrectomy as a uniformly successful operation. I have found it so. My friends have found it so. Their patients and mine are a happy crowd and are never tired of saying so. The unhappy ones are equally vocal, but they are so rare that they hardly cloud the picture. I have therefore come to believe that the bad results of gastrectomy are the results of bad gastrectomy. Recurrent ulceration at the stoma was seen in 9 out of 30 gastrectomies done by me in 1936 at a time when I believed that the pyloric antrum, since it secreted alkali, could safely be left; I have seen no instances in the 10 years before I advocated this heresy or in the 18 years since I abandoned it.

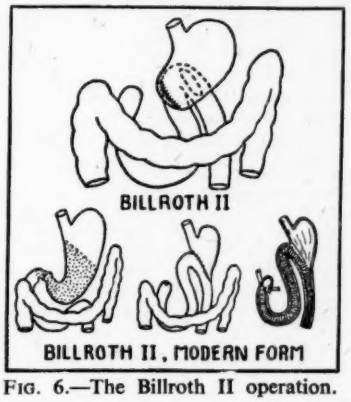


FIG. 6.—The Billroth II operation.

Dumping Syndrome

The dumping syndrome is in the news. Some centres speak of it as a matter of everyday occurrence, while others seldom see it, and such variation from clinic to clinic suggests that the cause is technical rather than inherent in the operation. So many disabilities are included under "dumping" that the more general term "post-gastrectomy syndrome" is preferable. Among the many possible causes are:

1. Operation on the wrong patients, on men with a small lesion of the duodenum and a large lesion of the mind. I have three times been persuaded against my better judgment to operate on such patients by physicians who had come to the end of their resources. In each case I regretted it. The first of these patients went on complaining till I lost sight of him at the beginning of the war. The second, a nurse, had a series of further operations (vagotomy, splanchnicectomy, and so on) by other surgeons, and finally died. The third, who had been "dumping" for two years, came running after me in Oxford Street a month ago, saying, "You will be glad to know I am perfectly well and can eat anything." "How did it happen?" I said. "Oh," he replied, "my mother died and I got a new job that I like."

FIG. 7.—The Billroth II operation; post-operative skiagram.

2. Too high gastrectomy. An operation that leaves only an inch or two of stomach on each curvature may be safe against further ulceration, but it is most unsuccessful functionally.

3. A full-width anastomosis—the old-fashioned Polya. This operation may have two unhappy sequelae; filling of the proximal loop giving a sense of postprandial distension, and stretching of the mesentery of the jejunum where it forms part of the stomach wall, with the inevitable symptoms of fainting, sweating, and nausea that are part of the vaso-vagal mesenteric reflex.

FIG. 8.—Two-stage gastrectomy for adherent duodenal ulcer. (Figs. 4 to 8 suggest a resection much more conservative than is actually performed, because the stomach elongates greatly when its vessels are ligated, and as the branches of the left gastro-epiploic artery are tied the fundus comes right away from the spleen. Estimates of the level of gastric resection by reference to the spleen are meaningless, because the size of the spleen and its attachments to the stomach are quite inconstant. The most disastrous mistake in peptic ulcer surgery, because it is irremediable, is to remove too much stomach, and unless the surgeon lifts up the left costal margin from time to time to see how much is left before he divides the stomach, he is apt to make this mistake. The size of a pint mug is right.)

4. Anastomosis anterior to the colon, an operation that is successful when the colon behaves but is entirely at the mercy of its vagaries.

5. Fixity of the stomach and the stoma owing to adhesions or to insufficient division of the vascular pedicles. The reduced stomach and the new stoma occupy quite different positions from the normal stomach and pylorus which they replace, but they should be just as free to distend and to empty, and to move about as they do so.

Conclusion

I believe that to-day, and till something better appears, the treatment of peptic ulceration that has proved resistant to medical treatment is by gastrectomy—Billroth I gastrectomy for gastric ulcer, Billroth II gastrectomy for duodenal ulcer. I know my own results till 1935. Since that time I have suffered the interruption of five years in the Army, the greater part overseas, and the destruction of my house with all my records. Secretaries, since my return, have been characterized by their impermanence, and those patients whom I should like to follow are scattered over four continents. I cannot therefore give accurate figures of my own, and, when asked to write on peptic ulceration for Butterworth's *Encyclopaedia*, I gave the aggregate figures of four surgeons whose work is well known to me.

Total number of operations	1,456
Death rate	2.7%
State of survivors:	
Satisfactory	93%
Better but not symptom-free	5.6%
Worse (including re-ulceration)	1.4%

The causes of death after gastrectomy are shock, infection, chest complications, and leakage from the duodenal stump. Shock has been eliminated by transfusion, infection by chemotherapy, chest complications by preparation before operation and early movement afterwards. Duodenal leakage does not happen to the surgeon who realizes that discretion is the better part of valour where it is the patient rather than himself that needs the valour. There should be no mortality, and there would be none if gastric surgeons were afforded the same privileges as are demanded as a right by their prima-donna colleagues—special theatres, new apparatus as soon as it is invented, theatre sisters who are left in the job long enough to become really expert, and anaesthetists and first assistants who work for years on the same team.

In 1935 my total mortality in operation for uncomplicated ulcer was 4.5%. In 1935 transfusion was used seldom and with caution, physiotherapy was reserved for elderly arthritics, and chemotherapy had not yet been invented. I do not know my mortality to-day, but I have certain pointers. No patient with a primary peptic ulcer has died in my wards at Guy's Hospital since 1938. Of my Service patients, only one has died, from sloughing of the stump of a Bancroft's operation, since which disaster I have discarded this device. Of 115 private patients operated on since my return to civil life, in Nuffield House and the Royal Masonic Hospital, one, a gentleman of 66, died on the second day from extensive mesenteric thrombosis. Compared with the dangers and disablements the lesion brings, these risks are small.

TREATMENT OF MASSIVE BLEEDING FROM PEPTIC ULCER

BY

A. G. OGILVIE, M.D., F.R.C.P.

N. CARDOE, M.B., B.S.

AND

F. H. BENTLEY, M.D., F.R.C.S

(From the Departments of Medicine and of Surgery, the Royal Victoria Infirmary, Newcastle-upon-Tyne; and the University of Durham)

In a previous report from the Royal Victoria Infirmary, Newcastle-upon-Tyne, Ogilvie and Spencer (1950) described the results of treatment, in a general medical clinic, of massive bleeding from gastric and duodenal ulceration. Among 170 consecutive patients admitted as emergencies because of severe bleeding the mortality was 2.4%. The series was in no way a selected one, and contained all such patients admitted to one medical clinic during the years 1941–8 in an industrial area, where the majority of severe medical and surgical emergencies find their way to the teaching hospital; 27 of the patients (16%) were aged 60 or over. It was recognized that there were many variables to consider, such as the age of the patient, the duration of severe bleeding before admission to hospital, the presence of a chronic ulcer, the situation of the ulcer, the cardiovascular condition of the patient, and the presence of intercurrent disease. Although the series was not a very large one, it did not appear from the analysis of the cases that any of these factors, except that of age, had seriously affected the results.

Since that report was published observation has been continued and developed in the hospital in order to define a policy in the treatment of massive ulcer bleeding, particularly in regard to the relationship of medical and surgical treatment, and to assess its precise results. Tanner (1951) explained clearly the need for such a policy in the management of this difficult problem if the results are to be properly evaluated, and described his own large experience following different methods of treatment (Table I). In his hands the procedure with the lowest mortality in a series of 854 patients was the Finsterer method, by which is meant: "To operate on all patients giving a history of chronic ulcer as soon as a diagnosis of massive bleeding is made. If it is uncertain whether the bleeding is due to chronic ulcer an

expectant policy is followed, but if bleeding is repeated an exploratory laparotomy should be carried out." This plan resulted in a mortality of 7.5%, but it should be noted that very many (43%) of the patients were over 60.

TABLE I.—*Tanner (1951): Peptic Ulcer and Gastritis Groups (Incidents of Bleeding)*

	No. of Cases	Over 60 (%)	Operated on (%)	Overall Mortality (%)	Average Age of Patients Who Died
Period No. 1, 1941 to 1943. Operation avoided	193	33	5	10	62
Period No. 2, December, 1943, to June, 1944. Operation earlier—always gastrectomy ..	60	33	15	20	62
Period No. 3, June, 1944, to December, 1947. Operation early—simplest to stop bleeding ..	312	35	11	11	65
Period No. 4, January, 1948, to September, 1949. "Finsterer" ..	215	43	59	7·5	65
Period No. 5, January to July, 1950. Selective surgery with bias towards conservative treatment	74	55 (34 over 70)	18	13·5	72
Totals ..	854	40		11	65 yrs.

Present Investigation

In our own hospital all patients with haematemesis and melaena are admitted as emergencies to the medical clinic on "reception" duty. There are four medical clinics, and each in rotation is on emergency duty three to four days at a time every two weeks. We have studied the cases of massive bleeding admitted to two out of the four medical clinics, and so obtained an adequate number of patients without the clinical problems of management becoming too unwieldy, and without creating any bias in selection of cases. The survey was continued from March 1, 1949, to November 30, 1951, a period of two years nine months; during this time 358 patients were admitted urgently to the two clinics because of severe ulcer-bleeding, representing rather more than half of the total admissions to the medical department for this condition during the period. The series does not include the 170 cases previously reported.

The policy of treatment agreed between the two medical clinics—those of Professor F. J. Nattrass and Dr. A. G. Ogilvie—and the surgical department, was first to treat by medical measures all patients admitted because of severe bleeding from peptic ulcer, and then to consider surgery (and generally to treat by operation) as soon as it was plain that medical treatment was not succeeding. The recognition of the lack of success of purely medical measures was usually made within 24 hours of admission if bleeding still continued; or at any time while the patient was in hospital if the bleeding, having once stopped, started again. Pursuit of this policy brought about 10% of our patients to operation: several surgeons were concerned in the series, but most of the operations were carried out by one surgical firm.

Our report is concerned with bleeding from peptic ulcer, a term which includes acute ulcers and multiple erosions. Those few cases of massive bleeding known to be due to other causes—notably gastric carcinoma and oesophageal varices—have been excluded from our figures. The evidence on which the diagnosis of ulcer depended for inclusion in this series of 358 patients is:

Ulcer demonstrated radiologically	169
Good clinical evidence of ulcer: barium meal negative	113
Ulcer found at operation or necropsy	57
Presumed acute ulcers (no pain; no evidence of liver or blood disease; barium meal negative or refused)	19
	358

We admire the persistence and skill of Avery Jones and his colleagues in securing so high a percentage of accurate diagnoses in the early days of the illness, but do not believe the ordinary medical clinic can emulate the special facilities and experience of these workers. With us the diagnosis has depended on clinical criteria, and on subsequent radiological examination (in most patients) when the acute emergency was over. Early in our series a patient who died of bleeding had had a previous radiological examination which suggested oesophageal varices; at necropsy the bleeding was found to be due to chronic peptic ulceration. Since that time, in any case of clinical doubt we have acted on the assumption that the haemorrhage was from a peptic ulcer, and in only two patients in two and a half years has the practice led to a known error in diagnosis: these two patients, with no previous dyspeptic history, were operated upon for uncontrolled bleeding, and were found to be suffering from hepatic cirrhosis. No harm came to them, and we have continued in the belief that when the diagnosis is in doubt the slight risk inherent in an exploratory operation in a patient with hepatic cirrhosis does not justify withholding operation from a patient who continues bleeding, although the source is uncertain.

Medical Treatment

To say that the first step in treatment is a careful and accurate history might seem like a repetition of the obvious, but it is emphasized here because the details of the history are important. The previous health, digestive history, source of haemorrhage, amount of blood lost, time of bleeding, and interval before admission are essential pieces of information, and are made clear only by careful inquiry. The first examination of the patient, on the other hand, though it must be adequate, need not be too detailed. Any but the very lightest palpation of the abdomen is to be avoided. Pallor, restlessness, sweating, disorientation, and dehydration are to be looked for, and can be readily ascertained by a general survey. The search for hypothetical masses in the abdomen can, and indeed should, be left for a later opportunity in most cases, though the possibility of an associated perforation must be excluded. The administration of morphine is a routine measure, and the patient lies flat in bed and is kept warm but is not artificially heated. The blood group is ascertained on admission, and the appropriate blood ordered if required. If bleeding has ceased the condition of the patient will be improving, and he may not require blood: in the presence, however, of any of the clinical signs of haemorrhage an immediate transfusion is given.

Indications for Transfusion

In making the first assessment of the patient on admission the pulse rate and blood pressure are not reliable guides, though at a later stage, when the patient is settled in bed and rested, the pulse rate is of the first importance in assessing progress. More certain immediate evidence of severe or continuing blood loss is to be obtained from noting the clinical signs of pallor, restlessness, or sweating. Crude information, too, can be secured from the level of haemoglobin. The presence of sweating or restlessness; anaemic pallor, as shown in the palms of the hands and in the conjunctivae; or a haemoglobin level of 60% or under are our indications for immediate transfusion, which is continued at an appropriate rate and may total three, four, or more litres of blood, until the condition of the patient, a haemoglobin level of at least 75%, and (at this later stage) the pulse rate indicate satisfactory progress. This is a particularly urgent matter in the elderly patient, in whom anoxia is a most serious complication; not only are there the dangers attendant on a reduced oxygen-carrying power of the blood, in particular coronary insufficiency, but there is also the potential danger of further haemorrhage in a person with a low blood level.

Whether the patient is transfused or not, careful and constant observation are needed, so that recurrence of

haemorrhage may be recognized promptly. We have found the half-hourly pulse chart to be the most helpful routine observation for this purpose, though daily haemoglobin estimation is useful also. Not only should the pulse rate remain steady, but it should gradually come down to less than 90 a minute if progress is to be accepted as adequate. A tendency to sweat about the head is often an early sign of renewed bleeding, and may be observed before signs of restlessness or dehydration are evident. Increased pallor is often obvious, but may be difficult to detect, especially in artificial light. If bleeding is slight but persistent there may be no other evidence than a fall in the haemoglobin level or a failure of the level to rise despite the giving of blood. These evidences of further bleeding are countered by further transfusion until the general condition of the patient, the haemoglobin level, and the pulse rate are reassuring, or failure to respond to transfusion indicates the need for surgical treatment. We deprecate the use of aspiration by stomach or duodenal tube as a means of observation, for it seems to us that the information which the method affords can be obtained by simple clinical observation.

Although the pulse rate will call attention to any sudden change, it has to be remembered that the pulse may be quickened from causes other than bleeding. We have not found that haemorrhage increases the respiration rate as a rule, so that if a rise in pulse rate is accompanied by an increase in respiration some cardiac or pulmonary cause should be considered, particularly if the clinical appearances do not suggest bleeding; otherwise blood may be given to a patient with early heart failure or bronchopneumonia who does not require blood and cannot tolerate it. A further caution in regard to transfusion exists in the relation between the anaemia of haemorrhage and heart failure. In elderly patients in whom moderate but repeated bleeding has been ignored or inadequately treated large transfusions are dangerous, and repeated small amounts are required.

Diet and Fluid

There has been much argument about the best dietary regime for patients after a haematemesis, but the more important question of the total fluid intake has been relatively neglected until recent years (Avery Jones, 1947). Except when blood is voided, vomiting is unusual, and if it should occur it may indicate a complication such as perforation or pyloric stenosis. The patients are usually thirsty, but do not always ask for fluid; some are too weak or too ill to drink adequately without help and encouragement. It is our practice to encourage patients to drink the contents of three 1-litre jugs of water each 24 hours, in addition to their two-hourly milk feeds, and most patients accept this volume of fluid readily. It is, however, essential that the nursing staff should become "fluid conscious." When fluid intake is adequate there is no difficulty about "water balance"; and when, in addition, enough blood has been given we have had no urgent biochemical disorders requiring treatment.

Within a few hours of admission feeding by mouth is begun. The diet consists of two-hourly or hourly feeds of milk, cereal with milk, egg and milk, "ovaltine," then porridge or "farola," and so on. This dietetic practice is a routine one, and is followed in all our cases until recovery allows a normal diet. Sometimes a patient who is experiencing active haemorrhage feels nauseated and may refuse a feed, but feeding by mouth is interrupted for no other reason. The fact or suspicion of active bleeding is not regarded as a reason for starving the patient.

Ascorbic acid, 500 mg., is given daily, but no other routine medicinal remedy, except that magnesium trisilicate with phenobarbitone and belladonna in the form of a powder is prescribed if the patient complains of pain, but not otherwise. Morphine may be required from time to time, but if pain is persistent the question arises whether some complication other than haemorrhage is present.

It is a rule that these patients should not be allowed to become unduly constipated. The prejudice against the use of enemata is hard to understand: straining at stool is not good for an ill person, especially one suffering from haemorrhage, and there may be much discomfort if the bowels are not emptied regularly. If a bowel action does not occur naturally on any day an enema is given the following morning. This practice also provides useful information about progress.

Decision for Surgery

The surgeon is asked to see any patient in whom bleeding does not appear to be satisfactorily controlled. He thus becomes acquainted with the particular problem, and takes part in the decision and in the pre-operative observation. If after 24 hours' adequate medical treatment in hospital there is evidence of continuing bleeding—as shown by renewed fainting and collapse, the vomiting of further recent blood, or a recurrence of severe melaena—a pulse rate remaining between 90 and 120, or failure of the haemoglobin level to rise, operative treatment is decided upon. Similarly, if the bleeding stops but starts again while the patient is still in hospital no further attempt is made to cure him medically, but surgical treatment is decided upon. Past experiences have shown us that most of the patients who died after gastro-duodenal bleeding were those who had recurrent bleeding in hospital. Over the years it has not been uncommon for us to watch patients in whom there were apparent contraindications to surgery bleed two, three, or more times, with several days in between haemorrhages, only to be obliged to operate in the end, and wishing that so much of the patient's resistance and reserves had not been wasted.

Delay in starting surgical treatment is bad for another reason—the loss which the community suffers from extravagant use of blood in the continued medical treatment of a patient who has, in fact, become a surgical responsibility. We believe there may be patients in this series, and in others, who have recovered without operation, but only at the cost of large amounts of blood which might have been saved by more prompt surgery. The usual amount of blood given in this series *after* a decision was made to operate was between 1 and 1.5 litres, and this includes that given at operation and in the recovery phase. The economics of blood transfusion is too little considered nowadays. Our insistence on early and sufficient transfusion is not due to any spendthrift attitude in the use of blood. It will often save those recurrent haemorrhages which use up more blood and may still end fatally, leaving many sad reflections—among them, that all the blood has been wasted in that it has failed of its purpose.

Pre-operative Treatment.—As soon as the decision is made to operate the rate of blood inflow is increased (if necessary) until there is reasonable evidence of a satisfactory blood volume and haemoglobin level. Although no figure may be regarded as a fixed one, it is desirable to have the pulse rate not more than 100–110, and the haemoglobin not below 70% at the start of the operation. During this period it is important to empty the stomach with a tube of adequate calibre; we use an oesophageal tube, size 12. No attempt is made to investigate or correct biochemical disturbances: the problem is the urgent one of bleeding.

Anaesthesia.—We have been fortunate in having the skilful collaboration of Professor E. A. Pask and his colleagues in the treatment of our patients. Professor Pask tells us that, beyond the usual anaesthetic measures necessary in the gentle management of an ill patient, the only special danger lies in the risk of reflux of blood into the trachea. Therefore not only is the stomach kept as empty as possible by means of the indwelling tube, but induction of the anaesthetic is carried out in such a way as to minimize the risk of regurgitation, and an intratracheal cuff-tube is introduced as early in anaesthesia as possible: this is much facilitated by limiting the morphine given in the pre-operative period. To avoid regurgitation at the end of operation, the surgeon introduces the suction end into the stomach shortly before

the gastric suture line is completed. We have found this a useful manœuvre in preventing reflux when the cuff-tube is withdrawn. A blood drip transfusion is maintained throughout the period of anaesthesia. It has been our experience that the condition of the patient at the end of the operation—as judged by pulse rate, blood pressure, and pulse volume—has not been worse than at the start.

Operation

The surgical problem is to stop the bleeding, and it is therefore necessary to work quickly, though gently, until the bleeding vessel is secured; thereafter only gentleness is important. The surgical approach can be discussed under three headings.

1. *Partial Gastrectomy.*—In the presence of a chronic peptic ulcer partial gastrectomy is the best treatment when it is possible, for it not only solves the immediate problem of bleeding but leaves the patient in a satisfactory condition for his future life. If the ulcer is in the stomach, partial gastrectomy is almost always possible, even in the presence of a large perforating ulcer involving liver or pancreas, using a Billroth I (Schoemaker) type of resection. For chronic duodenal ulcer a Polya gastrectomy is often feasible, even when the ulcer has perforated posteriorly to form a chronic cavity in the pancreas. Transverse division of the duodenum at the level of the ulcer quickly exposes the ulcer cavity: the bleeding vessel (often the gastro-duodenal artery) can then be under-run and the bleeding stopped. If the ulcer is within 2 to 3 cm. of the pylorus, freeing of a cuff of posterior duodenal wall and routine closure in two layers is effective. If, however, the ulcer cavity extends to the right of the line of the gastro-duodenal artery it is hazardous to dissect the posterior duodenal wall, and it is wiser to leave the ulcer *in situ* and to close the duodenal orifice by an inturning method (Bentley, 1952), which securely closes the duodenum without disturbing the dangerous region behind it.

2. *Opening the Viscus and Ligation of the Bleeding Vessel.*—Although partial gastrectomy is usually possible, conditions exist at operation for bleeding from chronic ulceration when the surgeon should do the least possible, contenting himself with stopping the bleeding and so saving life. Such conditions may arise from the violent nature of the haemorrhage or from the presence of a severely inflamed duodenum with extensive periduodenitis and formation of an inflammatory mass involving duodenum and surrounding viscera. Extragastric ligation of vessels is of little value because of the great number of branches reaching the stomach and duodenum, and the freely anastomosing plexus of large vessels in the submucosa (Bentley and Barlow, 1952). Incision of the anterior stomach or duodenal wall and direct exposure of the bleeding artery in the ulcer bed is the only swift and reliable attack in the circumstances. Subsequent repair of the anterior duodenal wall may, however, so narrow the duodenum as to necessitate a posterior gastro-enterostomy; but this is quickly done, and leaves for the future the problem of more adequate surgery when the patient has recovered from his acute emergency.

3. *When No Ulcer is Discovered.*—On rare occasions careful palpation of the stomach and duodenum, and search behind the first part of the duodenum, do not reveal any scarring or ulcer thickening, and a difficult decision has to be made. Incision of the stomach and duodenal wall and search inside the viscus is an unsatisfactory procedure, for it is impossible to search adequately all the large potential area of bleeding; and the slender prospect of a positive result does not justify the soiling and further loss of blood that the method entails. More routine use of pre-operative gastroscopy may be the best answer to this problem, but we have not so far used this technique. In the absence of evidence of portal hypertension at operation, and if the surgeon is sure on clinical grounds that the bleeding was gastro-duodenal, and if blood can be seen through the wall of the upper jejunum, we believe that a partial gastrectomy should be carried out. We have done this seven times in the present series, and in only one case has the bleeding continued after operation. Search of the specimens revealed one or more shallow ulcers, or multiple erosions.

It is an anxious decision for the surgeon and his attendant medical colleague to make, but in our judgment the danger from leaving things as they are and merely closing the abdomen is greater than is the danger of an unsuccessful partial gastrectomy.

Post-operative Management

The routine treatment after partial gastrectomy needs only slight variation in the patient with haemorrhage. Transfusion is stopped as soon as the blood level is restored, but the usual free drinking we permit after gastrectomy cannot be indulged because of the tendency to post-operative ileus in the presence of much blood in the small bowel. Glucose-saline by intravenous or subcutaneous hyalase drip is therefore needed for about two days, together with restricted fluids by mouth. Early ambulation and breathing exercises are especially important in these ill patients: our most recent elderly patient—aged 80—who had bled from a hole in the gastro-duodenal artery for which 10 litres of blood had been given pre-operatively and 1 litre post-operatively, perambulated with assistance round the ward on the third post-operative day and went home on the twelfth day. Such old ill patients require continuous stimulation away from recumbency and from lethargy—both mental and physical. Appropriate measures are taken to deal with any disturbance in serum chlorides, alkali reserve, or serum potassium during the first days of recovery, before feeding by mouth becomes free.

Results

The series consisted of 358 consecutive patients admitted because of severe bleeding from peptic ulcer; 106 of them (30%) were 60 or over. The result of the agreed policy of treatment—namely, to treat all cases medically in the first instance, and by surgery if bleeding continued after 24 hours in hospital or bleeding having once stopped started again—was that bleeding was satisfactorily halted in 339 of the 358 patients. Of the 358 patients, 35 came to surgical operation—that is, about 10% of the total. Nineteen patients died, a mortality of 5.3%. The figures are analysed in Tables II and III.

TABLE II.—*Results of the Policy—2 Years 9 Months (1949–51)*

Treatment	No.	No. Over 60 Years	Death	Average Age Deaths
Medical treatment alone ..	323	95		
Medical treatment followed by surgery	35	11		
Total	358	106 (30%)	19 (5%)	62 years

TABLE III.—*Analysis of the Series by Decades*

Age	No.	Deaths
Under 30	43	0
30–39	52	1
40–49	78	2
50–59	79	6
60–69	71	3
70 and over	35	7
Total	358	19

It will be appreciated that it was never in our minds to compare medical and surgical treatment. It is of greater value to consider the precise place of surgery in the treatment of those patients (about 10% in this series) who are not responding well to a medical regime, and to determine the stage at which operation should be performed. In our

view there is no subject in which the artificial barrier between physician and surgeon is more out of place than in the management of massive haemorrhage from peptic ulcer. The problem is a combined one, and it was because of this view that we sought to assess the results of the combined policy here reported.

A mortality of just over 5% is a reasonable justification of the policy and the methods of treatment employed, and we feel satisfied to continue this form of medical–surgical treatment of the bleeding ulcer patient. Consideration of the details of the patients who died (Table IV) shows, however, that the mortality rate could have been lower. In 8 of the 19 fatalities (Nos. 1–6, 15, and 16) death seems to have been truly inevitable, having regard to the condition of the patient at the time of admission to hospital or the nature of the lesion (No. 16). In two or three of the cases it may be that earlier recognition of the grave nature of the illness and transference to hospital might have had a favourable result, but these eight patients, of average age 72 years, are to us the inherent inevitable mortality of the series.

More profitable, if more humbling, is contemplation of the other 11 fatal cases. In four (Nos. 7, 8, 9, and 10) death occurred from continued bleeding, and the surgeon was not called. The four deaths did not result from the application of our policy, but rather from a failure to apply it correctly, owing to errors in judgment. They emphasize the importance of the practice of employing surgery as soon as it is plain that medical treatment is not succeeding. In one of these cases (No. 8) it is true there was reason to believe from previous *x*-ray examination that the patient was bleeding from oesophageal varices. Some patients with severe bleeding due to peptic ulcer do not give an "ulcer" history, and we feel sure that operation is indicated in every case of persistent upper alimentary haemorrhage unless very clear evidence of hepatic cirrhosis or grave blood disorder is present.

In Cases 11, 12, 13, and 14 transfusion seems to have been inadequate. Blood was given too little or too late, or too little and too late. No patient suffering from haemorrhage can be regarded as doing well whose pulse rate is still raised or whose haemoglobin level is below 70%. This is the more important the greater the age, but it is true of all. A low haemoglobin level hinders healing and so inclines to further haemorrhage; and, in patients with defective coronary circulation, anoxia of apparently moderate degree may precipitate acute cardiac insufficiency (as in Cases 12, 13, and 14) or predispose to coronary thrombosis. Blood must therefore be given promptly and in an amount sufficient to restore the haemoglobin level to normal or reasonably near to normal, though for obvious reasons its administration may have to be spread over several days.

Three patients who died after operation might have been saved. In two (Nos. 17 and 18) excessive use of intravenous fluids after operation probably aggravated the pulmonary oedema and pneumonia from which these patients died. Once operation is completed and bleeding has stopped,

TABLE IV.—*Statement of Deaths*

No.	Age	Sex	Ulcer	Details
			Medical Treatment Alone	
A. Death considered inevitable:				
1	89	F	Chronic duodenal ulcer	Advanced senility changes and mental confusion precluded surgery. Death from effects of bleeding
2	77	F	,, gastric ,,	Bleeding stopped. Patient became drowsy and then unconscious. Necropsy showed senility changes but no certain cause of death
3	76	M	,, ,, ,,	Bleeding and vomiting for three weeks before admission. Death a few hours after admission. Large perforation in stomach; peritonitis
4	71	M	,, duodenal ,,	Known to have vomited blood day before; moribund on admission. Death in half an hour
5	61	M	,, gastric ,,	Several bleeds during last year. Recent bleed 12 hours before admission. Death 6 hours after admission. Necropsy: extensive caseating tuberculosis both lungs; enormous posterior wall ulcer
6	55	M	Subacute gastric ulcers	No chronic indigestion. Recent bronchitis and one melaena. Necropsy: rheumatic endocarditis; pneumoconiosis, acute bronchiolitis. Two subacute gastric ulcers. Death thought to be related to pulmonary rather than alimentary disease
B. Failure to advise surgery:				
7	67	M	Two chronic pyloric ulcers	Two haemorrhages during week before admission. Two further bleedings over 5 days in hospital. Transfusion inadequate
8	57	F	Chronic gastric ulcer	Several bleedings in hospital over 9 days. Liver palpable. Treated in error as a case of portal hypertension
9	55	F	No necropsy performed	Continued small bleedings in hospital over 16 days
10	54	M	Chronic gastric ulcer	Continued small bleedings in hospital over 8 days. Acute bronchiolitis and emphysema
C. Inadequate transfusion:				
11	73	M	None found at necropsy	Several severe bleedings over 3 weeks in and out of hospital. Transfusion inadequate and surgery not considered; but necropsy showed no source of bleeding
12	56	F	Chronic gastric ulcer scar. Four acute gastric ulcers	Bleeding three hours before admission; haemoglobin 60%; transfusion started after 7 hours. Death suddenly as last of the first half-litre was being given, due to acute cardiac ischaemia
13	48	M	Chronic gastric ulcer	Bleeding 1 day before admission; haemoglobin 50%; 1 litre of blood given. Two days later haemoglobin 53%. Death suddenly after further 7 days, due to acute cardiac ischaemia
14	42	M	,, stomal ,,	Bleeding for 6 days before admission; haemoglobin 39%; 1 litre of blood given; further bleed two days later. Death from cardiac ischaemia
			Medical Treatment Followed by Surgery	
A. Death considered inevitable:				
15	70	M	Chronic duodenal ulcer	Bleeding 1 day before admission. Very ill for some days before admission, but no signs of a perforation. Continued deterioration in hospital. Operation, sealed-off perforation; subphrenic abscess; bleeding duodenal ulcer. Gastrectomy and drainage abscess. Death after 3 days
16	54	F	Acute gastric erosions	Bleeding 2 days before admission; continued bleeding after 24 hours in hospital. Operation, no ulcer mass: partial gastrectomy. Continued profuse bleeding. Death 2 days later. Necropsy: multiple acute erosions with haemorrhages throughout the mucosa of the gastric remnant
B. Excessive post-operative intravenous therapy:				
17	70	M	Chronic gastric ulcer	Bleeding 1 day before admission: continued bleeding after 24 hours in hospital. Gastrectomy. Death after 4 days, with pulmonary oedema and bronchopneumonia, probably associated with excessive intravenous fluid
18	67	M	,, duodenal ,,	Bleeding 1 day before admission: continued bleeding after 24 hours in hospital. Operation: profuse duodenal bleeding, only partially stopped. Death after 5 days, with pulmonary oedema, probably associated with excessive intravenous therapy
C. Surgical death:				
19	35	M	Chronic duodenal ulcer	Perforated ulcer 2 months before: extensive pulmonary tuberculosis Several bleedings in hospital over 10 days; operation evaded because of pulmonary condition. Finally, operation—gastrectomy. Death from injury to aberrant pancreatic duct entering first part of duodenum

further intravenous fluid therapy, especially saline, has to be employed with caution. The last patient (No. 19) died from a technical hazard not solved by the surgeon—the presence of an aberrant pancreatic duct opening into the back of the first part of the duodenum. This duct was not seen in the oedematous scar, and a pancreatic fistula resulted from which the patient ultimately died. The problem would have been avoided by not disturbing the ulcer base in the pancreas, and closing the duodenal orifice by an inturning method, as mentioned above.

It is no doubt impossible to achieve the ideal level of skill, but there are reasonable grounds for thinking that several of our fatalities need not have occurred if treatment had been more skilful. If that is so, the inherent mortality of our series would be a figure between 3 and 4%. If we could obtain this result in a comparable series of patients we would be satisfied that we had reached as near to the truly inevitable death rate as could be achieved in our present state of medical knowledge and methods.

One further observation is pertinent—whether patients with known chronic ulcers of the stomach or duodenum should be advised to continue on expectant medical treatment or be operated upon because of the risk of bleeding or of perforation. Of our 19 fatalities, 14 had deep chronic ulcers, some of enormous size. An ulcer which has extended through the muscle coats of the stomach or duodenum is no longer peptic in nature, but has become a callous ischaemic ulcer—ischaemic until it ulcerates into a neighbouring large blood vessel—and is not capable of natural sound healing. Against the continued ill-health which these ulcers represent, with the risk of perforation or bleeding, has to be set the risk and result of operation. The mortality of partial gastrectomy in a skilled organization is about 2%, and the prospect of a healthy digestion and social life following this operation is about 90%. Equivalent facts about medical treatment of chronic peptic ulcer to set against these figures are less certain, and are the subject of separate investigations now proceeding.

Summary

A policy in the treatment of massive bleeding from peptic ulcer has been defined and applied, particularly in regard to the relationship of medical and surgical treatment, and its results are assessed.

The policy was to treat such bleeding by medical measures in the first place, and by surgery as soon as it was plain that medical treatment was not succeeding.

The importance of early and adequate transfusion in medical treatment is stressed.

The decision for surgery was usually made within 24 hours of admission to hospital, if bleeding still continued ; or at any time while the patient was in hospital if the bleeding, having stopped, started again.

In this way 358 consecutive patients were treated, of whom 30% were aged 60 or over : 10% of the patients came to operation The overall mortality was 5.3%.

The fatalities are considered and improvements in methods discussed.

The results are held to justify the policy of treatment, readily applicable to the medical and surgical departments, in collaboration, of a general hospital.

REFERENCES

Bentley, F. H. (1952). *Brit. J. Surg.* In press.
—— and Barlow, T. E. (1952). *Modern Trends in Gastro-Enterology*, p. 309. Butterworth, London.
Jones, F. Avery (1947). *British Medical Journal*, 2, 441.
Ogilvie, A. G., and Spencer, I. O. B. (1950). Ibid., 2, 138.
Tanner, N. C. (1951). *Edinb. med. J.*, 58, 293.

THE MALARIA ERADICATION SCHEME IN MAURITIUS

BY

M. A. C. DOWLING, M.R.C.S., L.R.C.P., D.P.H. D.T.M.&H.

Officer in Charge, Malaria Eradication Scheme, Mauritius

Until recent years the chief method of malaria control consisted in the destruction of the larval stage of the anopheles mosquito by means of surface oiling, usually combined with the clearing, canalizing, or draining of its breeding-places. This method has been employed in Cyprus, and the success of the campaign there is well known. Shortly after the end of the 1939–45 war a malaria-control scheme, based on anti-adult measures and employing the residual insecticide D.D.T., was undertaken in British Guiana under the direction of Dr. Giglioli. The universal spraying of indoor resting-places proved to be very successful against the chief local vector, *Anopheles darlingi*, which, being predominantly a domestic species, was reduced almost to the point of elimination in the course of the next few years. Unfortunately, however, the ever-present risk of anopheline extension from the neighbouring territories made necessary the continued application of the antimalarial measures. In 1948, following upon the success of the British Guiana experiment, the Government of Mauritius, in conjunction with the Colonial Insecticides Committee, decided to sponsor a similar scheme in an attempt at the total eradication of malaria from the island. It was considered that, eradication once obtained, it would be possible to prevent the reintroduction of anopheles at the port or airfield. The high recurrent annual expenditure on malaria would then no longer be necessary.

Mauritius is a small island of some 720 square miles (1,864 km.²) with a population of almost half a million. Malaria was introduced in the middle of the last century, and the year 1867 saw a severe epidemic of the disease affecting chiefly the capital town of Port Louis, which was probably the point of introduction. In this town, with a population of 47,000, there were more than 6,000 deaths in a single month. The disease rapidly encircled the island, transmitted by the African vectors, *Anopheles funestus* and *A. gambiae*. The former appears to have been the chief carrier, and, by breeding vigorously all the year round, maintained the infection at a high level on the coast during the winter months of June–December. It was possible to find as many as 1,200 adults of this species in a single room on the coast—an unusually high density. *A. gambiae* probably played an important part in the transmission of the disease during the epidemic summer months.

Residual Spraying

In the first instance an attempt was made to eradicate the two vectors by residual spraying. This primary method was to be supplemented or superseded by other measures if these were considered necessary in the light of experience. A small team, selected in the United Kingdom, arrived in Mauritius in November, 1948, to direct the experiment and, by setting up scientific controls, to interpret the results. For purposes of administration the island was divided into six districts, each with a locally chosen team of 50 to 70 men under the direct supervision of a field officer. A thorough house survey had been ably carried out before the arrival

of the research team in Mauritius, and this, together with detailed sketch maps of the countryside, made it possible for work to begin as soon as the preliminary training of spraying staff had been completed. Three distinct zones were chosen, and a different insecticide formulation was used in each. By contrasting the three preparations used—D.D.T. technical in kerosene, and the 50% wettable powders of D.D.T. and B.H.C. (gammexane)—it was hoped to use in the later stages the one which proved most effective and economical.

Spraying began in the middle of January, 1949, and was everywhere completed by May. A large residential area on the central plateau of the island was omitted, as it was well protected by efficient antimalarial works. Hand-sprayers of the knapsack pattern were used, but for large buildings petrol-driven power-sprayers were found to be invaluable. All possible domestic resting-places for the anopheles adults were treated, including barns, churches, temples, and factories as well as the native dwelling-houses, outhouses, and cattle-sheds. Technical D.D.T. was mixed in bulk with kerosene to make a 5% solution and distributed to districts from the central depot. The two wettable powders were mixed at field stores to make a 5% suspension in water. The aim was to apply a surface deposit of 100–150 mg. of D.D.T. or B.H.C., and this was checked by continual chemical analysis of test papers and rigid checking by the supervisory staff. The amounts used for the first spraying were higher and more variable than was desired, but the variability diminished considerably as the spray gangs became better trained. Table I shows

TABLE I.—*Total Spraying Figures, 1949–50*

Spraying	Houses Sprayed	Rooms Sprayed	Population Directly Protected
Jan.-May, 1949	75,829	376,998	324,181
Sept.-Dec., 1949	58,184	275,554	231,824
Sept.-Dec., 1950	48,090	220,697	193,640
Total	182,103	873,249	—

briefly the total number of buildings treated during 1949–50. In the light of the results obtained it was found possible to reduce considerably the areas sprayed after the first treatment. The second spraying included a re-treatment of the B.H.C. zone with D.D.T., as the former insecticide was abandoned at the beginning of 1950 on economic grounds. In 1951 the Shell product "supona D," which is an oil-bound suspension of D.D.T. readily miscible with water, was introduced.

Difficulties Encountered

To make the work of the unit easier an ordinance was introduced in 1948 authorizing personnel to enter houses for the purpose of spraying or scientific control work. Certain difficulties were of course experienced, particularly in the early stages of the scheme. The custom of replastering the interior walls of houses immediately before the New Year, chiefly among the Indian community, presented a very serious obstacle, as the redecoration took place soon after spraying had been completed and at the beginning of the malaria epidemic season. A second difficulty was that house-spraying took place during the winter, and coincided with the gathering of the sugar crop. Many families made their way at dawn to the canefields, leaving their houses locked, to return at dusk after the day's work. These and many other minor obstacles were overcome, thanks to the assistance willingly given by Government officials, estate managers, and many private individuals who appreciated the value of the scheme to the health of the community. It has been the practice of spray gangs to cover all furniture and furnishings as thoroughly as possible to protect them from the spray. This procedure consumed a good deal of time, as the average house contained a formidable amount of bric-à-brac, but it was an important factor in maintaining the good will of the inhabitants.

So as to be able to interpret accurately the results of the residual spraying, a series of scientific controls were set up during the brief period between the arrival of the team and the beginning of the campaign. Laboratories were equipped and a staff of laboratory assistants and field mosquito boys were trained. Macgregor in 1924 had made a thorough survey of the mosquitoes of Mauritius (Macgregor, 1947), and Sippe and Twining in 1942–4 had prepared tables of spleen and parasite indices for towns and villages throughout the island at different seasons of the year (Sippe and Twining, 1946). Since that time, however, new and efficient antimalarial work had been carried out under the supervision of the Director of Medical Services, Dr. A. Rankine, and the crowded residential plateau in the middle of the island, together with the business centre of Port Louis, had been virtually freed from malaria. Throughout the greater part of the coast the disease continued undiminished all the year round, as shown in the preliminary survey in 1948.

Examination of Children

Four spleen and parasite surveys were carried out, and the figures given in Table II represent a mean for the whole island (including the healthy central plateau). It can be

TABLE II

Survey	No. of Children Examined	Spleen Rate %	Parasite Rate %
Before spraying (1948)	3,585	34·8	9·5
After first spraying (1949) ..	12,105	15·3	2·4
After second spraying (1950) ..	14,577	2·8	0·4
After third spraying (1951) ..	17,208	2·5	0·1

seen that a considerable reduction has taken place since the initial survey in 1948. Thick drops were taken from each child, Giemsa-stained, and examined at the unit laboratory for the presence of parasites (which in Mauritius included the three species *Plasmodium falciparum, P. malariae,* and *P. vivax*).

In an attempt to estimate the degree of malaria transmission among the protected population, a continuous survey was instituted late in 1949. In this infant survey blood slides were taken from children born in the coastal zone since the first spraying in their locality, a positive being accepted as evidence that the child had contracted the disease in spite of the treatment of his dwelling with residual insecticide. In all, more than 31,000 children were examined between September, 1949, and the end of March, 1952, and only 34 positive cases discovered, representing a parasite rate of 0.11%. Out of some 4,500 children examined during the epidemic first five months of 1951, only one positive case was found, suggesting that transmission of the disease is now of a very low order. The corresponding age group, 0–2 years, in the first spleen and parasite survey of 1948 gave a parasite index of 10.0%.

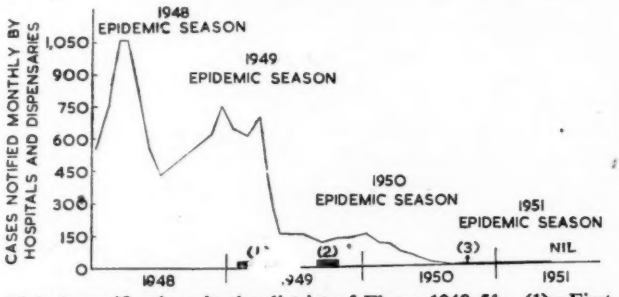
Notifications

In 1948 regular notification of all fresh cases of malaria treated at dispensaries and hospitals was introduced in Mauritius. Before this date no differentiation was made, and patients returned for treatment were counted as additional notifications. Thus the year 1948, a normal year for the disease in the island, makes a useful comparison with the years that follow. Table III shows the numbers of cases notified during the epidemic first six months of the years 1948–51.

TABLE III.—*Malaria Notifications During the First Six Months of the Years 1948–51*

	1948	1949	1950	1951
Total cases notified ..	29,938	17,742	3,762	852
Reduction % on 1948 figure	—	40·8%	87·4%	97·2%

Among those cases notified as malaria by dispensaries and hospitals during this period of 1951, 428, or just over 50%, had had blood slides taken before treatment and sent to the unit laboratory for examination. Of these slides only 26 were found to be positive. This suggests that many notified cases were not in fact malaria, but represented other forms of fever which, in a tropical country, may easily be confused with the disease. The district of Flacq, an area of about 115 square miles (298 km.²) with a population of 50,000, was originally a very grave malarious district. Furthermore, no antimalarial works had been introduced before 1948, so that Flacq has made a good experimental area for research. The graph demonstrates clearly how remarkable has been the reduction in the disease so far as can be judged from notifications. The infant survey was



Malaria notifications in the district of Flacq, 1948–51. (1)=First residual spraying with D.D.T. (2)=Second residual spraying with D.D.T. (3)=Beginning of pilot larviciding scheme. The "epidemic season" refers to the five-months period, January to May.

carried out in this district during February and March, 1951. Altogether, some 2,400 children aged between 3 months and 2 years were examined, and not a single positive case was found.

Results Reflected in the Death Rates

The vital statistics for the island indicate the influence of so striking a reduction in malaria on the general health of the community (Table IV). When considering the death

TABLE IV.—*Total Death Rates per 1,000 Mid-year Population, 1934–50*

Range in death rates, 1934–48	36·1–20·1
Mean death rate, 1934–48	27·2±3·37
Death rate, 1949	16·6
,, ,, 1950	13·9

rates for the whole colony it is more reliable to study those from all causes; the specific rates, such as the malaria mortality, do not represent an accurate index. The total rates also have the added advantage of reflecting both the direct and the indirect effects of the disease, and therefore demonstrate more closely the effects of its reduction.

The infant mortality rate has similarly undergone a significant reduction on the previous years, as can be seen in Table V. Improved health services, antenatal clinics, good

TABLE V.—*Infant Mortality per 1,000 Live Births, 1934–50*

Range in infant mortality, 1934–48	188·0–113·9
Mean infant mortality, 1934–48	150·0±19·2
Infant mortality, 1949	91·0
,, ,, 1950	76·4

years for the sugar crop, and other factors have undoubtedly contributed considerably to these reductions in mortality, but the virtual suppression of malaria must have played an important part in the process.

House-spraying

Study of the effects of house-spraying on the anopheline vectors revealed a very interesting situation. After the arrival of the team in Mauritius in November, 1948, a careful check was maintained on the mosquito population, particularly in the adult stage. Some 5,500 houses, scattered evenly throughout the island, were selected at random to form catching stations. Flitted regularly once a month by trained mosquito boys, these stations provided some evidence of the effect of the residual insecticide on the domestic vector species. Total figures for the catches of adult mosquitoes, by routine methods and also by regular "massed catching" by district staffs during the interspraying periods, are given in Table VI.

TABLE VI.—*Adult Mosquito Catches. Total Figures, 1948–50*

	No. of Houses Flitted	*A. gambiae*	*A. funestus*	Culicines	Vectors per House	Total Mosquitoes per House
Before spraying ..	7,817	2,486	31,496	113,235	4·35	18·8
After spraying:						
1949 ..	32,300	184	352	222,998	0·02	6·9
1950 ..	66,500	482	50	363,701	0·01	5·5

At the same time a continuous survey of anopheline breeding-places was carried out by whole-time skilled personnel. A review of the entomological data after the summer of 1950 revealed two facts of significance. First, *A. funestus* had been virtually eliminated, and, with the exception of two small sparsely populated areas on the coast, this previously very common mosquito could not be found either in adult or in larval form. In this connexion it is of interest to note that, during the whole of the period December, 1950, to March, 1952, no specimen of this species was discovered in spite of intensive searching. A similar effect has been observed on *Aëdes aegypti*.

The second point of interest was that *A. gambiae*, although apparently considerably reduced in numbers by residual spraying (98% reduction in 1950, according to the catches of adult mosquitoes in houses), was actually to be found breeding in large numbers throughout the coastal region in March and April of that year. In the course of a number of observations carried out to investigate this problem it was shown that *A. gambiae* could be found without difficulty in new unsprayed houses and cowsheds (as many as 98 adults of this species in a house and 306 in a cowshed), while the adjoining sprayed houses remained negative both to day- and to night-catching. Unfortunately, owing to lack of skilled personnel, it was not found possible to carry out more detailed research into the exact behaviour of *A. gambiae* under the conditions prevailing. This question is being investigated by an experienced entomologist, and the results may throw new light on the situation.

The problem which faced the organization at the end of the summer of 1950 was therefore briefly as follows. Residual spraying with D.D.T. as a single measure was sufficient, under conditions in Mauritius, to reduce malaria and its chief vector, *A. funestus*, to a very low level. The second vector mosquito, however, *A. gambiae*, although not apparently transmitting malaria to any extent, had shown itself capable of evading the lethal effect of the insecticide. It was therefore decided that measures additional to house-spraying should be undertaken in an attempt at the eradication of *A. gambiae*, and accordingly the second phase of the scheme was entered upon in October, 1950.

Pilot Larvicide Scheme

In Mauritius the winter season is relatively dry, and from September until the middle of December the breeding of *A. gambiae* is almost entirely restricted to permanent breeding-places along the coast. The plan was to carry out the wholesale destruction of larvae with Shell "malariol high-spread," in the first instance indiscriminately; and to follow this up by constant and detailed weekly checking, combined with "spot oiling" where vector larvae were discovered. It was hoped by this means to train personnel during dry months and at the same time to reduce considerably the population of *A. gambiae* when most vulnerable, so that it would be possible for the organization to cope with the great increase in work brought about by the advent of the rains in January. The district of Flacq, previously mentioned as a good experimental area, especially in that it possessed good natural boundaries which would help to

prevent anophelines from infiltrating from untreated areas, was selected for a pilot larvicide scheme. If the results in this district proved to be satisfactory it was decided to extend the scheme to the whole island in the winter of 1951.

At the end of the epidemic first five months of 1951 it was possible to conclude from results in Flacq that the use of the larvicide scheme on a larger scale would be warranted. Not only was breeding of *A. gambiae* reduced by more than 97% (as compared with other untreated areas of previously similar breeding intensity), but this mosquito remained confined to a narrow coastal strip throughout the summer and did not penetrate inland, as was previously the case during the rains. Accordingly, practice areas were set up in all the other districts for the training of personnel in the new methods, and the island-wide campaign began during the dry month of September, 1951.

Conclusion

Future policy must, of course, depend largely upon the reaction of *A. gambiae* to this second form of attack. The house-spraying, using a D.D.T. preparation, was repeated during the winter months of 1951, in the hope that the combined effect of the two methods would prove to be a potent weapon against this evasive species. However successful the scheme may be, it would be rash to relax vigilance for many years to come; for a small missed breeding-pocket might gradually re-establish transmission of the disease.

During the three years that the scheme has been in progress the total annual cost (including capital expenditure, running costs, salaries of United Kingdom and local personnel, and the laboratories with their equipment) has been the equivalent of a little over 3s. per head of population in Mauritius. It has been encouraging to see how rapidly house-spraying with a residual insecticide on a large scale can reduce the transmission of malaria in a community even when the vectors are as efficient carriers of the disease as *A. gambiae* and *A. funestus*.

NOTE.—Further information about the scheme, covering the period since this article was written, appears in a letter from the author on page 339.

I would like to express my thanks to Dr. A. Rankine, until recently Director of Medical Services in Mauritius, and to his successor, Dr. R. Lavoipierre; to Dr. P. C. C. Garnham, chairman of the Malaria Subcommittee of the Colonial Insecticides Committee; and to Mr. C. B. Symes, officer in charge of research to the Committee, for all their advice and assistance given during the course of the past three years.

BIBLIOGRAPHY

de Caires, P. F. (1949). *Mosquito Control Service, Annual Report, 1948.* British Guiana.
Dowling, M. A. C. (1951). *Nature, Lond.*, 167, 67.
—— *Malaria Eradication Scheme, Mauritius.* Corona. Vol. II, Dec., 1950, p. 452; and Vol. III, Jan., 1951, p. 26.
—— *Malaria Eradication Scheme, Annual Report, 1950.* Government of Mauritius, April, 1951.
Giglioli, G. (1948). *Malaria, Filariasis and Yellow Fever in British Guiana.* Mosquito Control Service, British Guiana.
Macgregor, M. E. (1927). *Mosquito Surveys.* Wellcome Bureau of Scientific Research, London.
Sippe, G., and Twining, M. (1946). *Survey and Field Treatment of Malaria in Mauritius.* Government of Mauritius.

Writing on the hospital services in *The Fortnightly* (July), Professor A. P. Thomson, dean of the faculty of medicine at Birmingham University, says: "We have in the Midlands some first-rate hospitals, but in 1948 the regional board took over from a county authority, and immediately closed, a hospital for chronic patients, of whom there were 16. There was no indoor sanitation, and the only water supply was from a stand-pipe in the yard. Ten of the patients were bedridden. That was the worst example, but there were several larger hospitals that were not much better. I was so astonished at what I saw that I got photographs of several of these places, and when I am reproached that hospital development is slow I get comfort from looking at the pictures, because we have nothing like them now."

THE SUPERIORITY OF "CAMOQUIN" OVER OTHER ANTIMALARIALS

BY

INDER SINGH, M.B., B.S., F.R.F.P.S., M.R.C.P.Ed.
Lieutenant-Colonel, I.A.M.C.

AND

T. S. KALYANUM, M.B., B.S.
Captain, I.A.M.C.

The rational objectives of antimalarial therapy have always been (1) to prevent deterioration in the patient's health by treating an attack vigorously and bringing it to a speedy end; (2) to prevent relapses; and (3) in the absence of world-wide mosquito control to reduce the patient's infectivity to the mosquito. Since the advent of chloroquine, proguanil ("paludrine"), and mepacrine ("quinacrine") the use of quinine has rapidly receded into the background. These three drugs have been very effective in eradicating M.T. malaria, although exceptions in the case of proguanil have been met with. None of them tried singly, however, cures B.T. malaria, but pentaquine and combinations of quinine and pamaquin, proguanil and pamaquin, and quinine and pentaquine have considerably lowered the relapse rate. In treating the acute attack mepacrine is often superior to proguanil, but both are inferior to chloroquine. In some respects "camoquin," which has been recently introduced, seems to be superior to chloroquine. We record here our observations on its use in comparison with some of the above drugs.

Clinical Trials

Only pure infections of M.T. and B.T. malaria were selected. The patients were a heterogeneous crowd of soldiers aged between 16 and 35. They had acquired recent infection after a variable stay in endemic and hyperendemic zones in various parts of India. None of them had been on suppressive treatment or had had any antimalarial drug before treatment was started. All of them were treated in hospital under identical conditions. They had the usual febrile forms of the disease, and criteria for relapse were an enlarged spleen, history of malaria within the previous two years, and the presence of gametocytes in the blood on the first day of the disease.

Antimalarial therapy was given only to those who showed almost persistent parasitaemia for 24 hours. Parasite counts were done, being expressed as the number of infected cells per 1,000 R.B.C.s. Haemoglobin estimation, red-cell counts, total and differential leucocyte counts, serum-bilirubin estimations, tests for serum methaemoglobin, and routine urine examinations were done, and blood pressure was recorded before starting treatment in every case. Temperature was recorded four-hourly. Therapy was always begun at the onset of a rigor, to ensure accuracy and uniformity. Blood smears were continued six-hourly for parasite counts till they became persistently negative for 72 hours. In order to record any evidence of immediate or delayed toxicity other blood and urine examinations, as stated above, were made after 48 hours and again 10 days later, after starting treatment. Blood pressure was recorded daily. In the case of mepacrine and proguanil these investigations were confined to recording the duration of pyrexia and the period of asexual parasitaemia. In the case of camoquin only, the patients were followed up for the last 14 months for evidence of relapse.

Results

The results are mostly summarized in Table I. Other points are included in the discussion to avoid repetition. As we had only five relapses in the camoquin series and none in the chloroquine series, we record the results obtained by various treatments, except in the camoquin series, in fresh cases only. The results obtained with proguanil have been difficult to predict, as they were somewhat erratic; those with mepacrine were steadier and better, but did not compare favourably with those obtained with chloroquine and camoquin. A combination of proguanil and quinine was better than a combination of proguanil and mepacrine or mepacrine alone.

Discussion

Pharmacology.—Camoquin is chemically 4-(3'-diethylamino-methyl-4'-hydroxyanilino)-7-chloroquinoline, and is sold as the dihydrochloride dihydrate. It is a light-yellow crystalline powder, slightly soluble in water, giving a 5% solution at room temperature. The drug is rapidly absorbed from the gastro-intestinal tract and adequate blood concentrations are obtained about an hour after oral administration. Its concentration in the erythrocytes is double that in the plasma, but is many more times in the spleen, liver, kidneys, lungs, brain, and heart, the highest being in the spleen. Adequate concentrations are maintained for a week or more after a single dose. The leucocytes may contain it for 22 days after its administration. There is little or no discoloration of the skin. The parent drug is not excreted in the urine, but 10 to 20% of the daily dose is excreted as a degradation product, and the elimination can be increased by acidifying the urine.

Animal Studies.—In animals a dose of 5 to 10 mg./kg. orally or 2.5 to 5 mg./kg. parenterally produces no acute or cumulative clinical or histopathological effects. In rats a dose of 800 mg./kg. orally is lethal, and doses between 65 and 195 mg./kg. affect growth. Monkeys tolerate 25 mg./kg. daily for a month. In therapeutic doses in man no toxic symptoms have been recorded. Continuous heavy doses, which will never be used therapeutically, have given rise to lack of energy, insomnia, epigastric discomfort, and anorexia. The drug has been found six times as effective as quinine against *Plasmodium cathemerium* in the canary (Gingrich), 8 to 25 times against *P. gallinaceum* in the chick (Coatney; Porter), and five times against *P. lophurae* in the duck (Marshall).

Clinical Investigations

In trophozoite-induced malaria, using the McCoy strain of *P. vivax* and the McClendon strain of *P. falciparum*, the curative dose has been found to be 350 to 400 mg. in B.T. malaria, and about three times this in M.T. malaria.

Results obtained by various investigators in India, the Philippines, the Amazon valley, and elsewhere have shown that the drug is very effective in the treatment of an acute attack and in diminishing the relapse rate (Penido *et al.*, 1948; Coggeshall, quoted by Payne *et al.*, 1949; Mein and Rosado, 1948; Simeons and Chhatre, 1947; Halawani *et al.*, 1947, 1948; Mein, Penido, and Deane, quoted by Ejercito and Duque, 1948; Chaudhuri and Chakravarty, 1948; Hoekenga, 1950). In most cases the drug terminated the attack and cleared the parasitaemia within 48 hours. The dosages were 1 g. six-hourly for three doses, or 1.2 g. daily for two days (Coggeshall), 0.25 g. 12-hourly for two doses (Halawani *et al.*, 1947), 0.5 g. single dose (Chaudhuri and Chakravarty, 1948), and 10 mg./kg. single dose (Simeons and Chhatre, 1947; Hoekenga, 1950).

Payne *et al.* (1949) obtained similar satisfactory results with two doses of 0.32 g. of the drug given intramuscularly at eight-hourly intervals without any local or systemic reaction.

In Table I the superiority of chloroquine over mepacrine and proguanil in terminating the attack and reducing parasitaemia is very significant. Similar results have been obtained by other workers using chloroquine against mepacrine and quinine (Most *et al.*, 1946), mepacrine (Pullman *et al.*, 1948), and proguanil (Canet, 1949) controls. Toxic symptoms have been rare with chloroquine, and a great advantage is that the treatment is completed in three days.

TABLE I.—*Comparative Results Obtained with Various Antimalarial Drugs*

Drug	Dose	Type of Malaria	No. of Cases	Duration of Fever in Hours			Duration of Asexual Parasitaemia in Hours		
				Av.	Max.	Min.	Av.	Max.	Min.
Camoquin	0·5 g. at onset of rigor and 6 hours later	B.T.F.	13	25	58	6	40	98	6
		B.T.R.	2	11	13	8	27	48	6
		M.T.F.	20	21	63	5	33	72	6
		M.T.R.	3	31	44	24	40	50	32
Chloroquine	1 g. initially, 0·5 g. 8, 24, and 48 hours later. Total 2·5 g.	B.T.F.	15	32	56	6	38	60	6
		M.T.F.	2	40	64	15	48	72	24
Proguanil	(*a*) 100 mg. t.d.s. for 10 days. Total 3 g. (*b*) 200 mg. t.d.s. for 10 days. Total 6 g.	B.T.F.	79	67	100	8	89	204	12
		M.T.F.	10	72	96	8	58	84	12
Mepacrine	200 mg. t.d.s. for 2 days and 100 mg. t.d.s. for next 5 days. Total 2·7 g.	B.T.F.	132	46	84	6	70	120	12
		M.T.F.	39	46	72	12	58	72	6
Proguanil and quinine	Proguanil 100 mg. and quinine 10 gr. (0·65 g.) t.d.s. for 2 days and proguanil 100 mg. t.d.s. for next 8 days	B.T.F.	15	37	72	6	36	84	6
		M.T.F.	2	22	30	14	30	54	6
Proguanil	Proguanil 100 mg. and mepacrine 100 mg. t.d.s. for 2 days and proguanil 100 mg. for next 8 days	B.T.F.	5	48	96	13	62	108	12
		M.T.F.	2	60	84	36	75	96	54

Duration of Fever

Chloroquine has now its rival in camoquin. Probably camoquin is the superior, but it is at present not readily available in the market, and a world-wide superiority has not yet been established for it. The period of treatment has been reduced to the minimum. A single dose is all that is required. The dosage and the cost are smaller than with chloroquine. In our series, although the maximum and minimum periods as regards duration of fever in B.T. malaria are almost identical with the two drugs, the average duration is in favour of camoquin, being 24 hours with camoquin and 32 hours with chloroquine, thus indicating that in a great number of cases fever is controlled earlier with camoquin. There is a slight difference in the rate of clearance of B.T. parasitaemia, the average period being 40 hours with camoquin and 38 hours with chloroquine. The minimum period is the same with both, but some cases have taken a day longer to clear up with camoquin. There has been, however, no relation between this and pyrexia, all the cases being afebrile during this period.

In M.T. malaria the average duration of fever with camoquin is about half that of chloroquine, although the maximum periods are almost identical. The minimum period is also less. M.T. parasitaemia also has cleared more rapidly with camoquin than with chloroquine, the average duration being 33 and 48 hours respectively. In M.T. malaria, therefore, camoquin seems to be definitely superior to chloroquine in controlling fever as well as parasitaemia.

Asexual Parasitaemia

When the last of the four samples of blood taken six-hourly before the administration of camoquin was negative it remained negative subsequently. It appears that erythrocytic forms do not develop from the exoerythrocytic forms when the blood contains camoquin. It would be interesting if this can be confirmed in *in vitro* cultures. It may be mentioned that only ring forms and amoeboid forms develop in culture under the influence of chloroquine. Failure of these early active forms to develop in numbers may account for the more rapid clearance of parasitaemia in camoquin-treated cases.

The rate of parasite clearance during the first 48 hours of treatment in M.T. or B.T. malaria was not found related to the initial parasite density. In fact some of the cases in which the density was greatest cleared up more rapidly than those in which the density was less. Presumably this indicates how powerful the drug is in its lethal action on asexual parasites. Schizonts did not develop in 12 out of 13 cases in which initially trophozoites were present.

In five cases the parasite density actually increased after the administration of camoquin, nearly doubling itself. In two of these the parasites almost completely disappeared within the next six hours. In the remaining three the increased density more or less persisted for 30 to 60 hours before rapid clearance. Clinically, however, there was no correlation between the duration of high density and fever, and the patients remained afebrile. The maximum parasite density in these cases was 11,000 and 17,000 per c.mm. of blood. We were unable to correlate this density with the concentration of the drug in the blood owing to lack of facilities. It is likely, however, that the delayed parasiticidal action may have resulted from a slower absorption of the drug from the gut. In such cases larger or parenteral doses would be more effective.

Sexual Parasitaemia

In cases in which gametocytes were initially absent before the administration of camoquin and remained so thereafter it may be concluded that asexual parasiticidal action was complete and any immature primary gametocytes which may have been present in the bone marrow or the spleen were entirely destroyed. Simeons and Chhatre (1947) obtained similar results in crescent-negative cases. They concluded that in this respect camoquin was superior to mepacrine. In crescent-negative cases mepacrine does not appear to have this occult gametocidal effect, although Rogan *et al.* (1944) found that in mepacrine-treated cases the incidence of crescents following one week after treatment was closely related to the mepacrine dosage, being less with larger doses.

In our series, where the gametocytes were already present in the blood, 90% cleared within six days on an average in B.T. cases, and about 60% during the same period in M.T. infections. The maximum period in one case of B.T. malaria was 14 days, and in a case of M.T. malaria 43 days. In Chaudhuri and Chakravarty's (1948) cases B.T. gametocytes disappeared in one to five days, counting from the day the drug was administered, while crescents took 7.8 days on an average to clear, the range being 1 to 26 days. Ejercito and Duque (1948) found that crescents persisted for four to nine days after treatment, but B.T. gametocytes showed marked reduction on the second and third days, and all disappeared by the fourth day.

Relapses

The use of heavy doses postpones the relapses. This was observed by Rogan *et al.* (1944) in mepacrine-treated cases, and Simeons and Chhatre (1947) obtained similar results with camoquin. With smaller doses M.T. cases relapsed more often than B.T. cases, as shown in Table II. These writers, however, have not clarified whether the cases treated had primary attacks or were relapsing cases, or whether they were pre-immunized subjects from endemic or hyper-endemic areas. The patients belonged to Kolhapur State Forces, in India, and were "mostly heavy infections."

TABLE II.—*Relation of Camoquin Dosage to Rate of Relapses (Simeons and Chhatre, 1947)*

Dose mg./kg.	Type of Malaria	No. of Cases	No. of Relapses	Latent Period in Days
10	B.T.	39	Nil	—
	M.T.	11	,,	—
7·5	B.T.	10	,,	—
	M.T.	6	2	20 each
5	B.T.	8	2	28 and 5
	M.T.	10	7	10 to 30

Ejercito and Duque (1948) treated both primary and relapsing B.T. and M.T. infections with camoquin, and obtained results as in Table III. Thus relapsing B.T. cases relapsed earlier and with about three times the frequency of primary B.T. cases. Primary M.T. cases did not relapse within six months, and were therefore apparently cured. A relapsing M.T. case relapsed after 81 days, a period longer than any of the B.T. cases. It may be noted that their dose was smaller.

TABLE III.—*Frequency of B.T. and M.T. Relapses (Ejercito and Duque, 1948)*

Dose	Type of Malaria	No. of Cases	No. of Relapses	Latent Period in Days
0·4 g., single dose	B.T.F.	40	2	39 and 56
	B.T.R.	44	6	25 to 38
	M.T.F.	22	Nil	—
	M.T.R.	22	1	81

During a period of observation of 5 to 12 months the relapse in *all* cases was 7.8%. Ejercito and Duque have compared it with a rate of 5% obtained by Mein, Penido, and Deane, in Brazil, with a single dose of 0.5 g. of camoquin. The latter claim that this dose "would cure 95% of all malaria cases (vivax and falciparum, whether primary or relapse)."

Hoekenga (1950), in Panama, treated 30 B.T. and 20 M.T. cases of malaria (unqualified) with a single dose of 10 mg./kg. of the drug and recorded only one B.T. relapse during a period of 6 to 16 months' observation.

With our dosage, which works up to 16–20 mg./kg., the relapse rate during a period of 14 to 16 months' observation has been as shown in Table IV.

TABLE IV.—*Relapse Rate in Present Series*

Dose	Type of Malaria	No. of Cases	No. of Relapses	Latent Period in Months
0·5 g., repeated in 6 hours. Total 1 g.	B.T.R.	2	Nil	—
	B.T.F.	13	2	10 to 12
	M.T.R.	3	Nil	—
	M.T.F.	20	,,	—

Making allowance for insufficient data in some of the above-quoted references and the comparatively small number of treated cases, one finds a marked diminution in the relapse rate with larger doses of camoquin. With one exception of M.T. relapse recorded by Ejercito and Duque (1948), the relapses, however, have all been in B.T. cases, although one would expect the opposite from the results of experiments with induced malaria (see above). The latter, however, explain to some extent why B.T. relapses are delayed and few. Apparently, therefore, the drug is superior in this respect to all other antimalarials and their various combinations. This is obvious from Table V, which gives results obtained by Most *et al.* (1946) in the treatment of *all* types of B.T. cases from the South-west Pacific zone.

Chloroquine definitely delays relapses, but after about four months it gives only slightly better results than mepacrine or quinine. A combination of quinine and pamaquin is definitely much superior. A combination of proguanil

TABLE V.—*Comparative Results with Various Antimalarials in Therapeutic Doses (Most et al., 1946)*

Drug	Latent Period in Days and Percentage of Relapses			
	30	40	50	120
Chloroquine ..	Nil	Less than 1%	11%	70%
Mepacrine ..	9%	28%	40%	80%
Quinine ..	54%	67%	72%	85%
Quinine and pamaquin ..	—	—	—	11%

and pamaquin has been found by Fairley (1946) in the same theatre almost to double the latent period obtained by a combination of quinine and pamaquin, although the percentage of relapses remained the same in both series. Also in the same theatre Pullman *et al.* (1948) found the relapse rate in severe B.T. infections to be four and a half times that in moderately severe infections with a combination of quinine and pentaquine, the superiority of which, therefore, still has to be proved.

Toxicity

Blood and urine examinations which were carried out showed no changes, and camoquin was well tolerated by all patients without giving rise to any complaint. Toxic symptoms with mepacrine and proguanil were also very rare. A few patients complained of abdominal pain after proguanil, and some vomited after mepacrine.

Summary

Camoquin has been found definitely superior to proguanil and mepacrine in the treatment of malaria. In M.T. malaria it seems to be superior to chloroquine in controlling fever and asexual parasitaemia. In B.T. malaria, fever is controlled earlier with camoquin, but in a few cases there is a slight difference in the rate of clearance of parasitaemia in favour of chloroquine. Evidence suggests that with larger doses of camoquin relapses are few, and are delayed. Although further experience is required to establish a world-wide superiority, especially over chloroquine, the use of a single dose and the smaller cost are distinct advantages.

We are indebted to Major S. P. Mukerji, I.A.M.C., for his kind help in the management and care of the chloroquine series of cases; to Messrs. Parke, Davis and Co., Bombay, for the free supply of camoquin; and to Lieutenant-General D. R. Thapar, Director-General, Armed Forces Medical Services (India), for his permission to publish this paper.

REFERENCES

Canet, J. (1949). *Pr. méd.*, **57**, 1015.
Chaudhuri, R. N., and Chakravarty, N. K. (1948). *Indian J. Malar.*, **2**, 115, 117.
Coatney, G. R. Malarial Report No. 365, Board for Coordination of Malarial Studies, O.S.R.D., Washington, D.C.
Ejercito, A., and Duque, M. (1948). *J. Philipp. med. Ass.*, **24**, 633.
Fairley, N. H. (1946). *Trans. roy. Soc. trop. Med. Hyg.*, **40**, 105
Gingrich, W. Malarial Report No. 365, Board for Coordination of Malarial Studies, O.S.R.D., Washington, D.C.
Halawani, A., Baz, I., and Morkos, F. (1947). *J. roy. Egypt. med. Ass.*, **30**, 99.
—— —— —— (1948). *Ann. trop. Med. Parasit.*, **42**, 304.
Hoekenga, M. T. (1950). *Amer. J. trop. Med.*, **30**, 63.
Marshall, E. K. Malarial Report No. 365, Board for Coordination of Malarial Studies, O.S.R.D., Washington, D.C.
Mein, R. M., and Rosado, P. N. S. (1948). *Rev. Serv. esp. Saúde públ., Rio de J.*, **1**, 1059.
Most, H., *et al.* (1946). *Amer. J. med. Sci.*, **212**, 550; *J. Amer. med. Ass.*, **131**, 963.
Payne, E. H., Sharp, E. A., and Nickel, K. C. (1949). *Amer J. trop. Med.*, **29**, 353.
Penido, H. M., de Souza, E. F., and Bezerra, F. P. G., jun. (1948). *Rev. Serv. esp. Saúde públ., Rio de J.*, **2**, 23.
Porter, R. J. Malarial Report No. 365, Board for Coordination of Malarial Studies, O.S.R.D., Washington, D.C.
Pullman, T. N., *et al.* (1948). *Proc. Cent. Soc. clin. Res.*, **19**, 83.
Rogan, J. M., *et al.* (1944). Report on Malaria Therapeutic Trial, Assam, Malaria Research Committee, General Headquarters, India.
Simeons, A. T. W., and Chhatre, K. D. (1947). *Indian Med. Gaz.*, **82**, 255.

OBSERVATIONS ON METHODS OF MEASURING THE ERYTHROCYTE SEDIMENTATION RATE

BY

A. GOLDBERG, M.B., F.R.F.P.S., M.R.C.P.

AND

HUGH CONWAY, M.B., B.Sc., M.R.C.P.

(From the Gardiner Institute of Medicine, the University of Glasgow, and the Western Infirmary, Glasgow)

The most popular methods of measuring the erythrocyte sedimentation rate (E.S.R.) are those described by Westergren (1921) and Wintrobe and Landsberg (1935); the latter is commonly known as the Wintrobe method. Technically, these procedures differ mainly in the nature of the anticoagulant and the length of the sedimentation tube; the bore of the tube is the same in both. Many investigators have compared the methods to assess their reliability, and Davis (1946), in a critical survey, concluded that the one of choice for general purposes is that of Westergren, because it is less sensitive to minor influences and more responsive when sedimentation is rapid. Recently, Gilmour and Sykes (1951) found, in a large series of cases, that a raised Wintrobe result was always accompanied by a raised Westergren; but a raised Westergren was not accompanied by a raised Wintrobe in 27% of female and 28% of male cases. They considered that the Westergren method is the more efficient as a pointer to organic disease.

In the course of routine haematological investigations we observed, within a short space of time, two patients in whom the E.S.R. by the Wintrobe method was normal whereas very high readings (over 100 mm. in one hour) were obtained by the Westergren. In both instances an abnormal protein was subsequently discovered in the blood, and if only the former method had been used an important diagnostic clue would have been lost. Because of these paradoxical results we decided to compare the two methods in a small series of cases. This investigation confirmed the confidence of Davis (1946) in the superiority of the Westergren method. It is, however, convenient to be able to perform a full haematological examination, including the E.S.R., on 5 ml. of oxalated blood as recommended by Wintrobe (1933). A series of experiments was therefore designed to explore the possibility of adapting the Westergren method, in which sodium citrate is the anticoagulant, to the use of oxalated blood.

Technical Methods

All needles and syringes were dry-heat sterilized and the sedimentation tubes were scrupulously cleaned and dried before use. Care was taken to ensure that the tubes were set in a strictly vertical position with air-bubbles excluded.

Blood samples for the Wintrobe and Westergren tests were taken by venepuncture. First, with the needle left *in situ*, 5 ml. of blood was delivered from a syringe into a glass bottle containing dry oxalate mixture (ammonium oxalate 6 mg., potassium oxalate 4 mg.). A second syringe containing 1 ml. of 3.8% sodium citrate was then attached to the needle and 4 ml. of blood withdrawn; this blood–citrate mixture was transferred to a clean dry test-tube. Both specimens were mixed by gentle shaking for approximately one minute. For straightforward testing the sedimentation tubes were charged immediately, the Wintrobe with the

TABLE I.—*Details of Cases in which the Westergren and Wintrobe Results were Dissimilar*

Case No.	Sex	Diagnosis	Initial E.S.R. (mm.)						Remarks
			Wintrobe			Westergren			
			½ hr.	1 hr.	2 hr.	½ hr.	1 hr.	2 hr.	
1	F	Myelomatosis	6	15	61	145	151	154	Reversal of A/G ratio. Rapid clumping of red cells in Westergren tube
2	F	Unknown	0	0–1	2	110	115	130	Abnormal protein of undetermined nature in peripheral blood
3	F	Myelomatosis	1	5	—	—	145	149	Plasma g'obulin 11·6 g. per 100 ml.
4	M	Tuberculous pericarditis	1	2	10	18	36	70	Plasma proteins normal
5	M	Myocardial infarction	1	10	21	10	19	28	,, ,, ,,
6	M	,, ,,	2	6	27	7	25	38	,, ,, ,,

blood–oxalate mixture and the Westergren with the blood–citrate. Readings were taken at half, one, and sometimes two hours.

Comparison of the Wintrobe and Westergren Methods

Tests by the Wintrobe and Westergren methods were carried out simultaneously on 52 patients suffering from a wide variety of medical complaints. Since the measured length of the Westergren tube is twice that of the Wintrobe, standards of comparison must inevitably be arbitrary. In most cases, however, the first-hour readings in both methods corresponded fairly closely, and they were classified into four groups—namely, normal, up to 10 mm. (Westergren), and 15 mm. (Wintrobe); slightly raised, normal to 30 mm.; moderately raised, 31 mm. to 50 mm.; and high, above 50 mm. The results by the two methods were thought to be either similar or dissimilar depending on whether they fell into the same or different groups. No correction was applied for low packed-cell-volume values; doubts about the wisdom of applying such a correction have recently been discussed by Terry (1950).

Similar Results

Forty-six cases gave similar first-hour results; these were normal in 12, slightly raised in 16, moderately raised in 9, and high in 9. In all 12 cases with normal results the Wintrobe and Westergren readings agreed closely. Of the 25 cases with slightly or moderately raised results, 22 gave Wintrobe readings higher than Westergren, whereas the opposite obtained in 7 of the 9 with high results.

Dissimilar Results

The results were dissimilar in six cases, normal Wintrobe being associated with raised Westergren readings. The discrepancy was gross in three cases, marked in one, and slight in two; brief details are shown in Table I. In all instances the Westergren result was more compatible with the clinical condition of the patient. The observations which originated the investigation were made in Cases 1 and 2.

Tests on the first four cases were repeated at intervals over a period of ten days, and the Westergren results never altered appreciably. The Wintrobe method, however, lacked this consistency, and in Cases 1, 2, and 4 the readings varied from test to test. In Case 2 there were actually gross fluctuations, for on some occasions, as in the initial test, sedimentation was negligible, whereas on others it was maximal in half an hour. The readings in Case 4 rose gradually until after nine days they were almost identical with the Westergren figures.

Influence of Anticoagulant and Tube.—To determine whether the anticoagulant or the sedimentation tube was primarily responsible for the dissimilarity in results, citrated and oxalated blood samples from Cases 1 and 4 were tested in both Wintrobe and Westergren tubes. The results, which are recorded in Table II, show that in both tubes the citrated blood sedimented more rapidly than the oxalated. In these cases, therefore, it was the anticoagulants and not the tubes that were decisive; the oxalate seemed to act as a retarding agent. In Case 1 the oxalated blood sample in the Wintrobe tube gave a first-hour reading, at the time of the experiment, of 50 mm., but sedimentation was still much delayed in the first half-hour compared with the citrate specimen.

TABLE II.—*Influence of the Anticoagulant and the Tube on the E.S.R. in Two Cases in Which the Classical Methods gave Dissimilar Results*

Case No.	Time (Hours)	Tube	E.S.R. (mm.)	
			Citrated Blood	Oxalated Blood
1	½	West.	134	6
		Wint.	75	6
	1	West.	140	20
		Wint.	76	50
	2	West.	142	60
		Wint.	77	65
4	½	West.	18	1
		Wint.	16	1
	1	West.	36	2
		Wint.	26	2
	2	West.	70	13
		Wint.	40	10

Use of Oxalated Blood in the Westergren Test

Blood, oxalated as for the Wintrobe test, was diluted with 3.8% sodium citrate in the usual proportion of 4 to 1; the E.S.R. was then measured in a Westergren tube. This procedure is referred to as the modified Westergren method.

Comparison of the Classical and Modified Westergren Methods. (a) Immediately after Withdrawal of Blood.—Thirty patients were studied. Citrated and oxalated blood samples were prepared in the usual way, and for the purpose of the modified test the latter were immediately diluted with citrate. The tubes were set up as soon as possible and the first-hour readings noted, that by the classical method being accepted as the correct sedimentation rate for each case. The results, which were classified into the same four groups as previously, are shown in detail in Table III. In general there

TABLE III.—*Comparison of E.S.R. by the Classical and Modified Westergren Methods*

	E.S.R. Group							
	Normal		Slightly Raised		Moderately Raised		High	
	a	*b*	*a*	*b*	*a*	*b*	*a*	*b*
E.S.R. (mm. at at 1 hr.)	2	2	20	15	38	34	104	117
	4	4	20	17	41	43	75	72
	8	10	25	25	37	37	62	65
	2	2	15	12	38	30	85	77
	2	2	28	30	40	15	80	65
	7	7			35	28	80	80
							136	140
							78	90
							88	103
							90	75
							65	79
							145	150
							130	130
Total cases ..	6		5		6		13	

a = the classical Westergren method. *b* = the modified Westergren method.

was fairly close agreement; the only serious discrepancy was in one case in the moderately raised group, when the readings varied from 40 mm. (classical method) to 15 mm. (modified method). Only one of the cases (Case 3) which gave

dissimilar results by the classical Westergren and Wintrobe methods was included in this series. Here the readings by the three methods, all performed at the same time, were 145 mm. (Westergren), 5 mm. (Wintrobe), and 150 mm. (modified Westergren).

(b) At Intervals after Withdrawal of Blood.—Ten further cases were studied by withdrawing from each (*a*) 25 ml. of blood, which was divided equally among five bottles containing dry oxalate mixture, and (*b*) 8 ml. of blood, which was added to a tube containing 2 ml. of 3.8% sodium citrate. After the usual gentle mixing 4 ml. of oxalated blood was extracted from one of the bottles and diluted with 1 ml. of citrate solution. This specimen was used for the modified test, which was set up immediately, and at the same time the classical test was performed on some of the citrated blood. The remaining four bottles of oxalated blood and the rest of the citrated blood were then stored at room temperature and the tests repeated after 3, 6, 12, and 24 hours. First-hour readings were recorded, and for comparison the correct E.S.R. for each case was taken as that obtained by the classical method performed immediately after the withdrawal of blood. Results were accepted as accurate only if the difference between the actual and correct readings did not exceed the following :

2 mm. when the correct E.S.R. fell within the normal range (0–10 mm.).
5 mm. when the correct E.S.R. fell within the slightly raised range (11–30 mm.).
10 mm. when the correct E.S.R. fell within the moderately raised range (31–50 mm.).
15 mm. when the correct E.S.R. fell within the high range (above 50 mm.).

The results (Table IV) show that the modified test maintained a higher degree of accuracy throughout the 24 hours than a classical one. After three hours' storage the former gave accurate readings in all ten cases, and even after 12 hours the results were still satisfactory in seven. As a final experiment the reliability of the modified test, after three hours' storage of oxalated blood, was further examined in ten additional cases. The previous findings were confirmed, for although the classical Westergren method was grossly inaccurate after three hours in five cases, the modified test gave accurate results both immediately and at three hours in all ten.

TABLE IV.—*Comparison of Classical and Modified Westergren Methods Immediately and at Intervals after Withdrawal of Blood*

	Time	Classical Westergren Method	Modified Westergren Method
No. of cases with accurate results	At 0 hours		10
	,, 3 ,,	9	10
	,, 6 ,,	5	8
	,, 12 ,,	1	7
	,, 24 ,,	0	2
Total cases		10	

The correct result for each case was the classical Westergren reading at 0 hours.

Discussion

The comparison of the Westergren and Wintrobe methods showed that the former was more reliable as a guide to the clinical condition of the patient ; this agreed with the findings of Gilmour and Sykes (1951).

In three cases (Cases 1, 2, and 3) of the six cases in which the Wintrobe method proved misleading because of false low results, the Westergren figures were very high (over 100 mm. in one hour). These gross discrepancies were constantly associated with an abnormality of the plasma proteins, and in Case 1, which alone of the three was investigated further, the retardation of sedimentation in the Wintrobe test appeared to be a function of the oxalate anticoagulant. A feature common to all three cases was rapid clumping of erythrocytes in the Westergren tubes ; this was immediately followed by speedy descent. Citrate solution has an inhibitory influence on rouleaux formation (Hesser, 1924), but when, as in the cases under discussion, the auto-agglutinating properties of the plasma are very strong this effect must be largely neutralized. Probably, however, it is not negligible, for the absence of any inhibition with the solid oxalate anticoagulant could explain the low Wintrobe readings on the basis of rouleaux formation being so rapid and massive as to interfere with the swift and orderly descent of the red cells. The development of small areas of clear plasma at intervals along the length of the Wintrobe tubes was a striking feature. An important point which is unexplained is the alteration in behaviour, from time to time, of the Wintrobe tests ; this was best exemplified by Case 2, in which on some occasions the reading was extremely low, whereas on others it was very high.

Although the cases which showed gross differences in results by the two methods all had abnormalities in plasma proteins, the converse did not hold, for others in the comparison series had a reversed albumin–globulin ratio and gave the expected high Wintrobe readings.

The modified test described in the second part of the investigation is both convenient and reliable, and therefore combines the main advantages of the classical Wintrobe and Westergren methods. It excels the former in accuracy, and is more convenient than the latter, for it can be performed, at the same time as other haematological examinations, on a single specimen of oxalated blood, which can be stored for a period of up to three hours before use. The test may therefore be useful in domiciliary practice when there must of necessity be a considerable time-lag before observations can be made on blood samples.

Just before this paper was submitted for publication, Goldberg, Glynn, and Bywaters (1952) reported preliminary observations on the simultaneous determination of the sedimentation rate by the Wintrobe and Westergren methods in patients with rheumatic disease. They found that in 10 out of 86 cases high Westergren were associated with normal or low Wintrobe readings.

Summary

The Westergren and Wintrobe methods of measuring the E.S.R. were compared in 52 patients suffering from a wide variety of complaints. In six cases raised Westergren readings, compatible with the clinical condition of the patients, were associated with normal Wintrobe readings.

This discrepancy between the two results was very gross in three patients, all of whom had an abnormality in plasma proteins.

A modified Westergren test which can be performed on oxalated blood after the addition of citrate is described.

When compared with the classical Westergren test, the modified test proved accurate in 49 out of 50 patients.

The modified test is reliable up to three hours after withdrawal of blood ; because of this and the convenience of performing all haematological examinations on a single specimen of oxalated blood it may be suitable for use in domiciliary practice.

We wish to thank Sir John McNee and Dr. J. A. W. McCluskie for advice and criticism in the preparation of this paper.

REFERENCES

Davis, L. J. (1946). *Practitioner*, **157**, 13.
Gilmour, D., and Sykes, A. J. (1951). *British Medical Journal*, **2**, 1496.
Goldberg, S., Glynn, L. E., and Bywaters, E. G. L. (1952). Ibid., **1**, 202.
Hesser, S. (1924). *Acta med. scand.*, Suppl. 9.
Terry, R. (1950). *British Medical Journal*, **2**, 1296.
Westergren, A. (1921). *Acta med. scand.*, **54**, 247.
Wintrobe, M. M. (1933). *Amer. J. med. Sci.*, **185**, 58.
—— and Landsberg, J. W. (1935). Ibid., **189**, 102.

SALM. TYPHI OSTEOMYELITIS

REPORT OF A CASE, WITH A REVIEW OF THE LITERATURE

BY

JOHN FERRIER, M.B., Ch.B., F.R.F.P.S.

Senior Registrar, Greenock Royal Infirmary

A case of osteomyelitis due to *Salmonella typhi* seen recently, demonstrating many of the characteristic features of that uncommon condition, has prompted publication of this review.

Case Report

A merchant seaman aged 22 was admitted to an isolation hospital on November 24, 1948, having had diarrhoea for four weeks. He was found to have typhoid fever. The urine and faeces yielded *Salm. typhi*, and the Widal reaction was positive. He had never had prophylactic inoculation, and the disease was severe. Chloramphenicol was not then available. By December his general condition had much improved, but aching pain had developed in the right middle finger and left upper thigh. However, these pains subsided, and he was discharged on January 20, 1949, in good general condition, the urine and faeces being negative on culture.

Painful swelling of the right middle finger recurred a few weeks later, and he was admitted to Greenock Royal Infirmary with a provisional diagnosis of typhoidal osteomyelitis. *X*-ray examinations showed a rounded cavity in the middle phalanx without periosteal reaction (Fig. 1). At operation the cavity was curetted, and pus yielded *Salm. typhi* on culture. Primary healing followed.

FIG. 1.—A focus of *Salm. typhi* osteomyelitis in middle phalanx of finger. Positive pus was recovered.

FIG. 2.—A focus of *Salm. typhi* osteomyelitis in mid-shaft of right tibia. This lesion subsided without pus formation.

FIG. 3.—A focus of *Salm. typhi* osteomyelitis at junction of middle and lower thirds of left radius. Positive pus was recovered.

On April 25, 1949, readmission was necessary because of severe aching pain in the right tibia, both great toes, right temporo-mandibular joint, upper right femur, and left seventh rib (see Fig. 4). There was high fever and collapse of the lower lobe of the left lung. *X*-ray examination of the affected bones was negative, except that in the right tibia a small rarefied area was seen in the middle third (Fig. 2). The spleen was not palpable and blood culture was negative. The condition resolved under treatment with sulphathiazole and penicillin, and all the bone foci subsided without pus formation.

On August 24, 1949, he was readmitted with a painful swelling of the left lower radius (Fig. 3). At operation a "collar-stud" abscess was opened and curetted. The pus again yielded *Salm. typhi*, and healing was again by first intention. On October 4 the Widal reaction showed *Salm. typhi* (H) agglutinated at 1 in 60 and no agglutination with *Salm. typhi* (O); on November 21 *Salm. typhi* (H) agglutinated at 1 in 240. By this time chloramphenicol had become available and a course of it was given, using the high dosage recommended by Smadel *et al.* (1949) designed to prevent relapses in acute typhoid fever: a total of 33.5 g. was given over a period of 19 days. The patient was seen again in December and a herniorrhaphy was carried out: he was then free of bone pains, and remained so until May, 1950, when a painful swelling developed on the right tibia but subsided in a week without pus formation.

At this stage the patient returned to sea and was not seen again until August, 1951, when he reported with a fluctuant swelling in the left temporal region of six months' duration (Fig. 4). A subperiosteal abscess was opened and curetted, the pus again yielding a pure culture of *Salm. typhi*. The organism showed a marked susceptibility, *in vitro*, to "aureomycin," and a course of that antibiotic was therefore given.

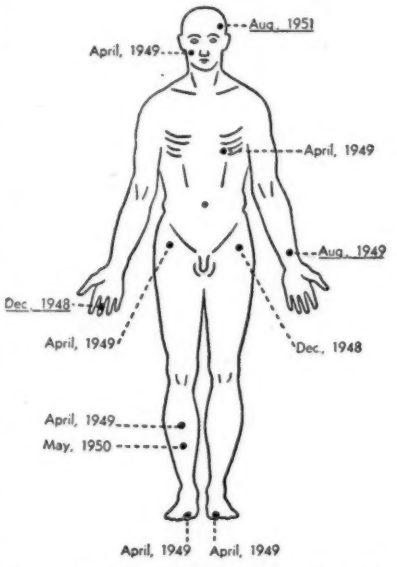


FIG. 4.—*Salm. typhi* osteomyelitis with multiple foci, showing sites and dates. The responsible organism was recovered at the sites underlined.

Review of Literature

Typhoidal osteomyelitis was first described by Maisonneuve (1835). After the discovery of *Salm. typhi* by Eberth in 1881, Ebermaier isolated the organism in pure culture from two cases of osteomyelitis. Chantimusse and Widal in 1887 recovered the organism from the marrow following inoculation into rabbits. Quincke and Stuhlen in 1894 recovered the organism from the marrow at necropsy in nine typhoid fever victims. The most comprehensive review of the subject is that of Winslow (1923).

Pathology and Clinical Features

In the initial bacteriaemia of typhoid fever bone-marrow cultures are often positive, and the value of marrow culture as a diagnostic measure has recently been confirmed by Hirsowitz and Cassel (1951), who obtained a positive result in 18 of 28 cases (64.3%). The marrow forms a suitable nidus for continued survival of the organisms in a dormant state, from which a recurrent bacteriaemia may at any time give rise to osteomyelitis.

In view of the frequency of positive marrow cultures it is surprising that the incidence of osteomyelitis after typhoid infection is so low. Thus Murphy (1916) found an incidence of only 0.82% in 18,000 cases, and Winslow (1923) a total of only 101 proved cases. This low incidence is even more surprising when it is remembered that until recently no specific treatment was available for typhoid fever. It is possible that the true incidence may be higher than usually supposed, and that some cases escape notice because the onset is delayed for years after the enteric infection, which may have been mild, forgotten, or not diagnosed as such, or because the clinician, in the case of osteomyelitis, accepts a laboratory report of "coliform organisms" without further investigation. This last point is well brought out in the case reported by Lever and Barber (1945). On the other hand, it is possible that some cases described before the present century may, owing to inadequate bacteriological methods, have been wrongly attributed to typhoid.

The onset of osteomyelitis is, as in the present case, usually in convalescence, and of the reported cases only

about 25% occur later. The disease is, however, well known for its tendency to appear for the first time many years after the enteric infection. Striking examples of this include those of Tubby and Hicks (1913), 13 years; Brock (1935), 14 years; Wardle (1935), 10 years; Lever and Barber (1945), 33 years; and van Dyk (1933), 24 years.

There is no doubt that injury is an important localizing and precipitating factor, particularly in cases in which the onset of bone involvement is delayed for years after the initial infection. The cases of Wardle (blow by hockey-stick), van Dyk (kick by horse), and Lever and Barber (repeated head injuries from low roof) demonstrate this. A bone operation may likewise be a precipitating factor, as in the case reported by Schein (1942). Ritter and Bochner (1936) described two cases (husband and wife) infected from the same source at the same time, both of which developed bone lesions. There is, however, no real evidence to suggest that a particular strain of organism accounts for the cases with bone involvement.

The disease affects mainly the long bones. Thus of 101 cases the tibia was involved in 56, the femur in 27, the ribs and costal cartilages in 27, and the skull in 11 (Winslow, 1923). The present case shows the tendency of the disease to attack the long bones, ribs, and skull, but involvement of the phalanges has also been a noticeable feature, and this appears to be only the third recorded case of involvement of the phalanx. Multiple foci occurred in this case, but are found in only 5% of cases (Veal, 1939).

The diaphysis is affected rather than the metaphysis, and the process begins under the periosteum, which becomes thickened and raised by sero-sanguineous fluid or pus. The process may subside or spread through the cortical bone of the medulla, forming a "collar-stud" abscess, partly within and partly without the bone, as was found on two occasions in this case. Sequestration is not an obvious feature, and sequestra are always very small (Winslow, 1923). Microscopically the pus may be full of small sequestrated spicules (Veal, 1939). When the skull is involved a Pott's "puffy tumour" with extradural abscess may form, as in the case of Lever and Barber (1945).

After adequate surgical treatment sinus formation is rare, but when it does occur it is noteworthy that the organism may persist in pure culture over a prolonged period (Winslow, 1923). The patient with a persistent sinus is a danger to the public health, and Bigelow (1912) reported that three people were infected from a sinus. It is also possible that, in the recurrent bacteriaemia postulated in this disease, organisms may also be excreted in the urine.

The severity of the osteomyelitic process is very variable, although normally slight; it bears no relation to the original enteric infection. Fatalities are rare and have been reported mainly in Continental literature up to 1916. Winslow (1923) mentions that the periostitis may result in formation of exostoses, and that persistent bradycardia may occur. Neither of these was noticeable in the present case.

Treatment

Keen (1898) stated that surgical excision and closure without drainage, as against the drainage and packing methods then used in ordinary osteomyelitis, was the treatment of choice; and despite its antiquity that statement remains accurate. First-intention healing was obtained on three occasions in the present case, and also in the cases of Brock (1935) and Ritter and Bochner (1936) in rib and costal cartilage, and of Wardle (1935) in the tibia. Primary healing is especially desirable in view of the risk to the public health of a persistent discharge.

The typhoid spine is reported to clear up with prolonged immobilization (Veal, 1939), and the same author rightly insists on the necessity for primary closure of the infected joint.

In the past, treatment by vaccines, both stock and autogenous (Garr, 1927), and by bacteriophage, both locally and intravenously (MacNeal, 1932), have been used.

The above case is one of the few, so far published, treated by chloramphenicol. Good results from this antibiotic have been claimed in the U.S.A. by Morse and Geiser (1950), in France by Barcat *et al.* (1950), and in this country by Gilles (1951) and Morgan (1951).

The present case relapsed after seven months, while Morgan's case has remained healed for a year. Morse and Geiser followed their case for only five months before reporting it. In view of the known natural history of the disease, and the relapses in the present case, it is unlikely that any of these cases are permanently cured. Gilles and Morgan both used chloramphenicol locally, and believe that healing of the wound was assisted. In the present case primary healing was obtained by surgery. Much should not be expected of chloramphenicol in this disease, since an apparently adequate course does not always prevent relapses in acute enteric fever (Douglas, 1950), and failures are usual in chronic biliary carriers (Woodward *et al.*, 1950). It appears clear that while chloramphenicol is helpful in dealing with the individual bone focus it is not curative, and its importance will lie in the more effective treatment of the initial enteric infection.

In the present case there have been no biliary symptoms, the faeces have been repeatedly negative, and cholecystectomy has not been advised.

Summary

A case of *Salm. typhi* osteomyelitis, with multiple foci, showing many of the characteristic features of typhoid is recorded and the relevant literature reviewed.

Adequate surgical treatment of the individual focus remains the only effective treatment. Chloramphenicol failed to prevent a recurrence.

I am indebted to Mr. A. Lyall, Mr. G. R. Thomson, and Dr. Andrew Love for permission to publish this case.

REFERENCES

Barcat, J., *et al.* (1950). *Mem. Acad. Chir.*, **76**, 412.
Bigelow, E. B. (1912). *J. Amer. med. Ass.*, **58**, 1339.
Brock, R. C. (1935). *Brit. J. Surg.*, **23**, 231.
Douglas, A. D. M. (1950). *Lancet*, **1**, 858.
Garr, C. C. (1927). *Sth. med. J.*, **20**, 296.
Gilles, H. M. (1951). *British Medical Journal*, **2**, 218.
Hirsowitz, L., and Cassel, R. (1951). Ibid., **1**, 862.
Keen, W. W. (1898). *Surgical Complications and Sequels of Typhoid Fever.* Saunders, Philadelphia.
Lever, J. M., and Barber, G. B. (1945). *British Medical Journal*, **2**, 459.
MacNeal, W. J. (1932). *Amer. J. med. Sci.*, **184**, 805.
Maisonneuve, J. G. T. (1835). Thesis, Paris.
Morgan, J. D. (1951). *Lancet*, **2**, 911.
Morse, C. W., and Geiser, F. M. (1950). *Arch. intern. Med.*, **85**, 280.
Murphy, J. B. (1916). *Surg. Gynec. Obstet.*, **23**, 119.
Ritter, H. H., and Bochner, S. J. (1936). *N.Y. St. J. Med.*, **39**, 877.
Schein, A. J. (1942). *J. Mt Sinai Hosp.*, **9**, 154.
Smadel, J. E., *et al.* (1949). *J. Amer. med. Ass.*, **141**, 129.
Tubby, A. H., and Hicks, J. A. B. (1913). *Lancet*, **1**, 304.
van Dyk, C. G. L. (1933). *S. Afr. med. J.*, **7**, 287.
Veal, J. R. (1939). *Amer. J. Surg.*, **43**, 594.
Wardle, E. N. (1935). *Clin. J.*, **64**, 121.
Winslow, N. (1923). *Ann. Surg.*, **77**, 319.
Woodward, T. E., *et al.* (1950). *J. clin. Invest.*, **29**, 87.

Continued record breaking in athletics, and not least in this year's Olympic Games at Helsinki, has prompted *World Sports*, the journal of the British Olympic Association, to persuade Professor A. V. Hill, F.R.S., to answer the question "Where is the limit?" in its August issue. In what he calls speculations, Professor Hill discusses whether there are physiological and anatomical limits to faster running and higher jumping, and begins by declaring that the human race is not improving, and it is very doubtful if the present generation of middle- and long-distance runners have a better supply of oxygen to their muscles than the best of their fathers and grandfathers. The secret lies in the efficiency with which muscular energy is used. The greatest efficiency is to run at a constant speed, and unfortunately high speeds spend proportionately much more energy than low ones. Thus in running a mile in four minutes a man who could cover each quarter-mile in exactly 60 seconds would use 4% less energy than a man who lapped 57, 63, 67, and 53 seconds per quarter-mile.

PARATYPHOID B OSTEOMYELITIS OF FRONTAL BONE

BY

E. PEYSER, M.D.

AND

M. MADORSKY, M.D.

(From the Departments of Neurosurgery and Clinical Bacteriology of the Hebrew University Hadassah Medical School, Jerusalem, Israel)

Although paratyphoid osteomyelitis has been described repeatedly in the literature we could find no case in which the skull bones were involved. Cases of typhoid infection of the skull, however, have been recorded by Winslow (1923), and by Lever and Barker (1945). Typhoid osteomyelitis occurs in almost 1% of typhoid infections, while the incidence of this complication is much lower in cases of paratyphoid fever (Winslow, 1923 ; Keith and Keith, 1926 ; Rozansky, Ehrenfeld, and Matoth, 1948).

Our observation of paratyphoid B osteomyelitis of the frontal bone deals with a hitherto unreported localization of paratyphoid osteomyelitis and is added to the 23 cases of bone involvement collected by Rozansky, Ehrenfeld, and Matoth (1948), and the cases of Ecker, Kuehn, and Recroft (1942), Ancherson (1947), Abrams and Gaenslen (1948), and Ludwig (1949).

Case Report

A man aged 54 was seen at a surgical out-patient clinic on April 27, 1951, because of a soft swelling at the right side of his forehead. There was no fever, pain, or any other subjective complaints. As *x*-ray examination of the skull showed foci of bone destruction in the right frontal area, he was referred to the neurosurgical department. Thirty-three years previously he had been ill with a "typhoid-like intestinal infection," after which he had not been well for almost a year. Since then he had been complaining of a dull pain in both legs which had been diagnosed as a rheumatoid condition. Seven years before admission he had had a swelling on the forehead similar to the present one ; it had ruptured after a blow on the frontal region ; a few millilitres of pus was expelled and the abscess healed.

He was a fairly well nourished man. His temperature and pulse were normal. A small scar in the middle of the forehead pointed to the site of the previous abscess. At the right frontal area a soft fluctuating swelling, the size of a plum, was attached to the underlying bone as well as to the skin above. There was painless thickening of the lower third of the left tibia, the adjacent soft tissues being normal. General physical examination revealed no other abnormality. The sedimentation rate was 4 hours 10 minutes (Linzenmeier). A blood count showed: red cells, 5,140,000 ; haemoglobin, 80% ; white cells, 9,600 (polymorphonuclear neutrophils 75%, eosinophils 1%, lymphocytes 16%, monocytes 8%). Wassermann and Kahn reactions in blood were negative.

Radiographs showed several areas of bone destruction in the right frontal region, with irregular borders without signs of bone reaction. *X*-ray examination of the chest showed thickening and increased density in the posterior part of the left seventh rib, and thickening of bone in the lower thirds of both tibiae, with scattered areas of rarefaction and increased radiolucency. The cerebrospinal fluid, which was clear and under normal pressure (110 mm.), contained 72 mg. of glucose per 100 ml., 12 mg. of protein per 100 ml., and no cells ; Wassermann and Kahn reactions negative : gold-sol test, 000000. Aspiration of the frontal swelling yielded greyish-yellow pus ; no micro-organisms were found in direct smear or culture. Osteomyelitis of unknown origin, probably specific, with formation of subcutaneous abscess, was diagnosed.

On May 7, 1951, the diseased bone was freed by a curved incision at the right frontal region. A small amount of pus was found under the skin over an area of destruction of the outer lamina of the bone. The diseased bone was removed as a whole, and its borders were nibbled away with rongeurs until healthy bone tissue was encountered. On the inner lamina there were several holes of different sizes due to bone destruction. A fair amount of pus was found in the epidural space and evacuated. The dura mater was thickened with adherent granulation tissue, which was scraped off. Nowhere was this membrane destroyed. After removal of all affected bone a defect of 6 by 8 cm. resulted. Galea and skin were sutured individually. The operative wound healed by first intention, and the patient made an uneventful recovery, leaving hospital on the sixth post-operative day. He has been well since.

Microscopical examination of the removed bone showed chronic and acute osteomyelitis. Gram-negative bacilli were found in a direct smear of the pus evacuated during operation. By culture and biochemical reactions they were identified as *Salmonella typhi* B. Agglutination tests performed with the patient's serum gave the following results: *Salm. typhi* 1:100± ; *Salm. paratyphi* B 1:100±. Agglutination of the strain cultured from the patient's pus by the patient's serum 1:200++. Repeated cultures of the urine, stools, and duodenal juice were negative for salmonella organisms.

Comment.—This patient had recurrent osteomyelitis of the frontal bone 26 and 33 years after an enteric infection. Although no bacteriological and clinical data of this are available, there is no doubt that the micro-organisms cultured from the osteomyelitic pus—*Salm. paratyphi* B—were of the same type responsible for the primary disease.

We must further assume that the *x*-ray changes found in both tibiae and in one rib were also due to an invasion by the same micro-organism.

Discussion

Paratyphoid bacilli are known to stay in the bone marrow long after they have disappeared from blood, urine, and faeces (Ling *et al.*, 1940 ; Macdonald, 1941). Obviously they also can remain in the diploe of the skull.

Several cases have been reported in which the bone infection became manifest many years after the original intestinal disease (Jensen and Kock, 1913, 20 years ; Ecker, Kuehn, and Recroft, 1942, 24 years ; Ancherson, 1947, 18 years ; Rozansky, Ehrenfeld, and Matoth, 1948, 32 years). Most authors agree that trauma, physical strain, or an intercurrent disease may lead to activation of the clinically latent micro-organisms and produce the condition of acute osteitis and osteomyelitis. No such factors could be elicited in our own case.

Summary

A case of bacteriologically verified paratyphoid B osteomyelitis of the frontal bone is presented. Operation brought about complete recovery. Other bone lesions in the same patient (both tibiae, and one rib) are thought to be of the same origin. The frontal-bone lesion first appeared 26 years after the acute enteric infection, and recurred seven years later.

Incidence of bone complications in paratyphoid infection is rare, and this is the first case to be recorded in which the skull was involved.

We are indebted to Dr. A. J. Beller, head of the Department of Neurosurgery, to Dr. J. Gurevitch, head of the Department of Clinical Bacteriology, and the *X*-ray Institute of our hospital (head: Professor A. Druckmann) for their help.

REFERENCES

Abrams, R. C., and Gaenslen, F. G. (1948). *Nav. med. Bull., Wash.*, **48**, 306.
Ancherson, P. (1947). *Nord. Med.*, **36**, 2019.
Ecker, E. E., Kuehn, A. O., and Recroft, E. W. (1942). *J. Amer. med. Ass.*, **118**, 1296.
Jensen and Kock (1913). In W. Kolle and A. Wassermann's *Handbuch der Mikroorganismen*, **3**, 1054. Jena.
Keith, D. Y., and Keith, J. P. (1926). *J. Amer. med. Ass.*, **87**, 2145.
Lever, J. M., and Barker, G. B. (1945). *British Medical Journal*, **2**, 459.
Ling, C. C., Taur, S. S., Hsueh, P. C., and Yang, S. Y. (1940). *Chin. med. J.*, **57**, 11.
Ludwig, F. E. (1949). *Surgery*, **26**, 139.
Macdonald, A. (1941). *Lancet*, **1**, 174.
Rozansky, R., Ehrenfeld, E. N., and Matoth, Y. (1948). *British Medical Journal*, **2**, 297.
Winslow, N. (1923). *Ann. Surg.*, **77**, 319.

APPARATUS FOR DEMONSTRATING HAZARDS OF STATIC ELECTRICITY IN OPERATING THEATRES

BY

K. C. MAGEE, M.I.Mar.E.

Group Engineer, Southend-on-Sea Hospital Management Committee, General Hospital, Rochford, Essex

AND

A. BRACKEN, Ph.D., B.Sc., F.R.I.C.

British Oxygen Co. Ltd.

We were fortunate enough to be able to witness the remarkably effective demonstration of the explosive properties of mixtures of cyclopropane and oxygen given by Dr. H. J. V. Morton at the Royal Society of Medicine on December 1, 1950 (*Lancet*, 1950; *British Medical Journal*, 1951). The apparatus described here was devised as a result of this stimulus. It gives a startling demonstration of the ease with which static electricity may be generated and caused to produce an explosion with ordinary equipment used in operating theatres. The apparatus reproduces the condition that could arise in an explosive atmosphere when two bulky items, either equipment or personnel, are brought together or near enough to each other to produce a spark. Some such apparatus may possibly have been used already in laboratory studies (cf. Bulgin *et al.*, 1949), but, if so, it does not appear to have been described in the literature.

One form of the apparatus is shown in Fig. 1, and a photograph of an actual explosion involving cyclopropane and oxygen is shown in Fig. 2.

Description of Apparatus

The apparatus consists essentially of two electrodes sealed by non-conductive material inside a "cellophane" chamber. The points of the electrodes are set about ¼ in. (3 mm.) apart, and they project above a circular "perspex" block 2¼ in. (5.7 cm.) in diameter by ½ in. (1.3 cm.) thick, sealed on the perspex base. To fill the chamber with gas an entry port is provided from the outer edge of the main base to the inlet port in the centre of the block. A bronze ring is made to fit firmly on to the block, which is slightly tapered to provide a firm seating. The cellophane explosion chamber is prepared by cutting out a circle about 5 in. (12.7 cm.) in diameter from a sheet of thin cellophane, and, after forming it into a roughly hemispherical shape, placing it over the edges of the block. The bronze ring is now pressed down firmly, trapping the cellophane between it and the tapered edge of the circular block, producing a gas-tight joint. A flexible explosion chamber is thus produced with a rupture pressure of 100 to 150 lb. per sq. in. (10.54 kg. per cm.^{2}), which is also transparent and permits the spark to be observed in the event of a non-explosive mixture being present within the chamber. The flexibility of the cellophane allows the volumes of gases admitted to be varied within wide limits, and they can be estimated accurately enough for demonstration purposes.

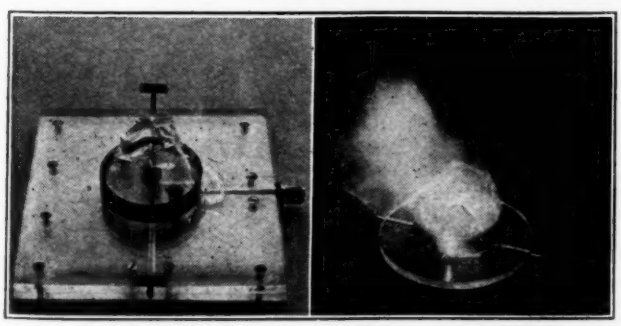


FIG. 1 FIG. 2

FIG. 1.—One form of the explosion chamber. FIG. 2.—Photograph of an explosion involving cyclopropane and oxygen, using another form of the explosion chamber.

A still simpler apparatus may be constructed from 2-in. (5-cm.) ebonite rod through which three holes are drilled, and the cellophane bag secured by adhesive tape.

Method of Demonstration

To introduce the explosive mixture the cellophane is first compressed over the electrodes to expel the air. The bag is then inflated by applying the outlet valve of a cyclopropane cylinder to the gas entry port of the apparatus. Acetylene, coal gas, or other inflammable gas may also be used. The cellophane bag is now half compressed, expelling half the cyclopropane. Oxygen is then used to fill the chamber to capacity. It now contains a 50% mixture of gas and oxygen. It is sealed by means of the screw plug provided for the gas entry port. The chamber retains the gas mixture for several minutes before leakage takes place.

An ether–oxygen mixture is readily introduced by passing oxygen through or over the ether in the bottle of an anaesthetic machine and thence to the gas entry port. A preformed gas mixture may also be introduced if desired.

One of the electrodes is now connected to earth and the other to an anaesthetist's trolley. Before making this connexion it is essential to neutralize any charges which may be present on the trolley by connecting it momentarily to the earth lead and thus eliminating any risk of pre-ignition. The safe minimum distance a person may be from the explosion is 10 ft. (3 m.) in a room or chamber of not less than 10,000 cu. ft. (283 m.^{3}). Care should be taken to observe this point when demonstrating. Light-gauge fuse wire is suitable for the leads. They are clearly shown in Fig. 2.

The trolley should stand on a non-conductive floor, preferably in a warm dry room, and should be provided with a rubber sheet and a dry blanket. On removal of the blanket a loud explosion takes place. The flash is orange in tint with cyclopropane and white with acetylene, ether having an intermediate colour. The explosion is due to the static charge induced in the trolley by the movement of the blanket on the rubber sheet. Measurements taken with an electrometer show the potential to vary from 10,000 volts to 15,000 volts when the blanket is removed by slipping it sideways on the surface of the rubber. Lifting the blanket vertically induces a smaller potential of 2,500 volts. The minimum potential so far recorded as producing an explosion with this particular apparatus is 2,700 volts.

To prevent loss of charge between the point of induction and the discharge electrodes, no earthed body or any mass which would serve as a capacitor should be near the conductors on the live side of the chamber. Care should also be taken to prevent tracking of the current in the explosion chamber itself. This can well be caused by a film of moisture left by a previous explosion or residing on the surface of the perspex. This can largely be pre-

vented by arranging that the explosion chamber is slightly warmer than the atmosphere in which it has to operate.

Variants of the Procedure

Some interesting variants can be made. For example, the earth lead from the explosion chamber may be held by an insulated person or it may pass to an insulated anaesthetic machine. In each case an explosion takes place on removal of the blanket from the trolley. This may be explained by the capacity of the "earth" being large enough to cause the difference in potential between the initial system (the trolley alone) and the final system (the trolley and "earth") to exceed 2,700 volts.

The following demonstration is also of interest. One of the leads from the gas-filled explosion chamber is picked up by an assistant who is to act as the "earth." This lead should be about 10 ft. (3 m.) long. The other lead, of similar length, is left free. The operator, wearing insulated rubber boots, now connects himself electrically to the trolley and charges it (and himself) by removing the blanket. He feels no sensation while this is carried out. The operator may now break his contact with the trolley, walk several steps across the room, and then, by taking up the free lead from the explosion chamber, produce an explosion by apparently mysterious means.

The subject of the dangers of static electricity has recently received attention in a Ministry of Health Memorandum (1951), and the demonstrations described here lend force to the need for observing the precautions suggested in the memorandum.

It may be of interest to note that the explosion photograph (Fig. 2) was taken in a large darkened room under the conditions described here. The light emitted during the explosion was enough to produce the photographic image.

We wish to express our thanks to the Southend-on-Sea Hospital Management Committee and the Directors of the British Oxygen Company Ltd. for permission to publish this communication. We also thank Mr. T. R. Mumby, engineer-in-charge, and Mr. J. Wood, medical photographer, both of Southend-on-Sea Hospital, for their co-operation in this work.

REFERENCES

British Medical Journal, 1951, 1, 298.
Bulgin, D., Llewellyn, F. J., and Thomas, G. (1949). *Lancet*, 1, 789.
Lancet, 1950, 2, 754.
Ministry of Health Memorandum No. 191 (Med.) (1951). *British Medical Journal*, 2, 1324.

Medical Memoranda

Transmesenteric Herniation

Transmesenteric herniae are amongst the less common causes of intestinal obstruction and, in consequence, the literature on the subject is usually in the form of isolated case reports. Treves (1885) originally drew attention to the occurrence of defects in the terminal mesentery, and considered that the ovoid circular defects were congenital whilst the rare slit-like type of hole was traumatic in origin. Cutler had personal experience of two cases, both in young children, and after review of 46 reported cases concluded that most of these defects were of developmental origin. Dolton (1944), after one personal case and a review, expressed the opinion that the condition should be regarded as congenital.

The traumatic theory was advocated by Brown (1920), who in his case recorded the history of a heavy fall and at operation the discovery of a slit-like mesenteric defect. Long (1927) added his support to the traumatic theory, having dealt with a 4-day-old child, with a pressure deformity of the thorax, in whom intestinal obstruction had resulted from herniation through an apparently recent mesenteric defect.

Inflammation with subsequent atrophy creating the defect has been advanced as a possible cause, and King (1935), from two cases, expressed the opinion that appendicitis explained the high incidence in the ileo-caecal region; in one of his cases there was an acute appendicitis with the tip of the appendix adherent to a knuckle of ileum which had prolapsed through a defect in the mesentery. It has been suggested that other inflammations might give rise to the same result, and Poponschik is quoted as having described a patient with tuberculosis of ileum and caecum involving the adjacent mesentery in which transmesenteric herniation of bowel took place through a defect in the involved segment of mesentery.

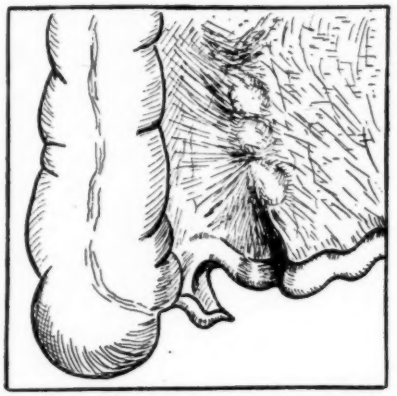


FIG. 1.—Sketch showing mesenteric adenitis with tenting, as suggested in the text.

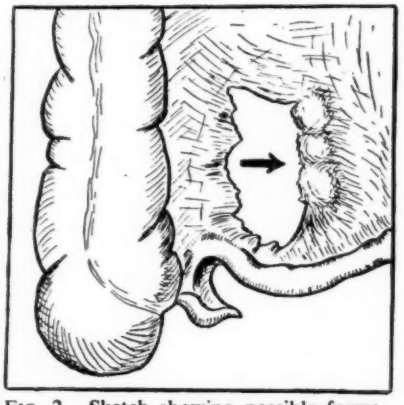


FIG. 2.—Sketch showing possible formation of the hiatus by the tearing of a stretched fibrosed mesentery.

In 1938 I recorded a case of internal strangulation through an aperture in the mesentery of a 33-year-old woman. This hiatus was ovoid in shape, had smooth margins, and lay between the ileo-colic artery and the lowest mesenteric branch. In the absence of local disease, I concluded that the defect was of congenital origin. Recently I encountered a further case of internal herniation through a mesenteric hiatus, and on this occasion there was ample evidence to support the theory that this defect was not of the congenital variety; in fact I now feel doubtful about other cases hitherto labelled congenital.

CASE REPORT

A man aged 28 had been quite free from complaint until five days before his admission to hospital, when he had had colicky abdominal pains without tenderness, guarding, or distension. After a short respite on medicine the pains returned in considerable severity and vomiting occurred. On the day before admission the colicky pains reached their maximum intensity and there was added generalized constant abdominal pain. From the onset of the illness there had been no bowel action or passage of flatus. One week before, whilst moving a heavy weight, he had felt something pull inside his abdomen, and the next day he had been conscious of a vague ache in the lower right abdomen. Some 10 years previously he had been troubled with odd attacks of pain in the right iliac fossa, and at this time the possibility of appendicular disease was considered.

A man of spare build, he lay quietly in bed on admission, and in spite of considerable abdominal distension showed little sign of distress. His temperature was 99.2° F. (37.3° C.), pulse 122, and respirations 22. The tongue was coated and the breath heavy. The vomitus was dark, foul-smelling, and profuse. In addition to the general abdominal distension there was uniform moderate tenderness and guarding. Dullness was present in the flanks. Although peristaltic sounds could be heard infrequently, they were of the high-

pitched tinkling variety; transmitted cardiac sounds were audible over the whole abdomen. It was possible to elicit splashing in distended loops of bowel. On rectal examination the pelvic peritoneum was found to be tender. The remainder of the examination was negative.

At operation it was found that several feet of small bowel had herniated from above through a defect in the terminal mesentery, and the weight of the prolapsed closed loops had in turn obstructed that part of the terminal ileum which formed the lower boundary of the hiatus. The viability of the loops was not in doubt, and reduction was affected without difficulty. It was then found that the caecum had undergone rotation and had accompanied the small bowel through the defect. After returning the caecum to its normal position it was seen to fill through the ileo-caecal valve. The appendix, which was normal in appearance, was not removed. It was noted that the mesenteric defect was ovoid in shape and about 3 in. (7.5 cm.) in length. The margins were rigid and smooth; the medial margin was swollen by the presence of a chain of enlarged glands suggestive of tuberculous adenitis. The congestion and oedema surrounding the defect obscured the main vessels. From the lateral side it was possible to bring across a longitudinal fold of peritoneum and attach it to the rigid medial margin of the defect with interrupted thread sutures, and thus the mesenteric hiatus was completely occluded. Several months after operation progress had been entirely satisfactory.

COMMENT

There is little doubt that in this case tuberculous mesenteric adenitis played an all-important part. There were vague pains in the right lower abdomen up to the age of 18, and, although at this time appendicular disease was suspected, the probability is that the real cause of the discomfort was mesenteric adenitis. It may be suggested that adhesions formed between the glands and the adjacent terminal mesentery and produced a tenting. The recent strain on a tented, fibrosed, and atrophied mesentery would be sufficient to tear an opening which was soon enlarged by the herniation of small bowel. Within five days of the strain the patient had his first symptom of obstruction, and operation was carried out five days later; in 10 days the margins of the defect would no longer be expected to present features of a recent tear.

The strongest supporter of the developmental theory would find it hard to attribute the case described to congenital causes. One must accept a congenital cause for mesenteric defects encountered in young people, but it is difficult to believe that patients with large defects can reach adult life without internal herniation and obstruction supervening. The defect encountered in the previously reported case was attributed to congenital causes, but, on looking back and in the light of the recent case, one cannot be so certain. In a hurried operation on a critically ill patient with distended coils of small bowel it would be quite easy to miss evidence of previous inflammation, if any such evidence remained. It is always interesting to note what few local signs persist several months after an appendix abscess has resolved, and therefore it would not be unreasonable to assume that an inflammatory condition in the right iliac fossa could eventually produce a mesenteric defect without showing, at the time of operation for the acute obstruction, any evidence of such inflammation. The fact that the ileo-caecal region is the common site for both acute and chronic inflammatory conditions would account for the predominant incidence of mesenteric defects in the terminal mesentery.

JOHN A. BATY, F.R.C.S.Ed.

BIBLIOGRAPHY

Baty, J. A. (1938). *British Medical Journal*, **1**, 671.
Brown, H. P. (1920). *Ann. Surg.*, **72**, 516.
Cutler, G. D., and Scott, H. W. (1944). *Surg. Gynec. Obstet.*, **79**, 509.
Dolton, E. G. (1944). *Brit. J. Surg.*, **31**, 275.
Jarvis, J. F. (1946). *Med. Pr.*, **216**, 123.
King, E. S. J. (1935). *Brit. J. Surg.*, **22**, 504.
Long, E. R. (1927). *Trans. Chicago path. Soc.*, **12**, 335.
Price, E. W. (1946). *Med. Pr.*, **216**, 211.
Smith, R. (1945). *Brit. J. Surg.*, **33**, 187.
Treves, Sir F. (1885). *British Medical Journal*, **1**, 470.
Wright, D. (1941). *J. roy. Army med. Cps*, **77**, 164.

Transmesenteric Herniation Due to Tuberculous Mesenteric Adenitis

My colleague, John Baty, recently read a paper on the subject of transmesenteric hernia at a meeting of the Shrewsbury Clinical Club, and described the case published above. I have since encountered a further case in which the defect was certainly due to tuberculous mesenteric adenitis.

CASE REPORT

A man aged 31 was admitted at 3 a.m. on September 11, 1951, from a sanatorium where he had been a patient on account of extensive bilateral fibroid pulmonary tuberculosis with some cavitation. Owing to his severe deafness it was difficult to elicit the history, but he admitted only slight occasional indigestion before the gradual onset at 10 p.m. on September 10 of a dull epigastric ache associated with nausea. The pain gradually became severe, and he had vomited thrice. The bowels had acted the previous morning. On admission there was some tenderness and guarding in his upper abdomen, but no rigidity and no distension. The pain did not appear to be colicky, and it was thought that the patient was unlikely to be suffering from either intestinal obstruction or a perforated ulcer. In view of the advanced pulmonary disease it was considered unwise to open the abdomen except for most definite indications. After 100 mg. of pethidine he at first appeared to be improving and did not vomit again; but the pulse rate tended to rise, and by the afternoon of September 11 there was a resonant suprapubic swelling typical of strangulated bowel.

A right lower paramedian incision, made under local analgesia, revealed blood-stained peritoneal exudate and gangrenous small intestine, but this could not be satisfactorily examined until general anaesthesia was induced by Dr. M. J. Harker with thiopentone, gallamine triethiodide, gas, oxygen, and trichlorethylene. The appearances at first suggested strangulation by a band or volvulus associated with a band, but when the whole affected area was delivered it was seen that there were two large loops of gangrenous bowel, one on each side of the mesentery, evidently communicating through a small defect near the attachment of the intestine (see illustration). We were immediately reminded of Baty's attribution of the mesenteric defect in his case to tuberculous adenitis, but at the time of operation we could not find any enlarged lymph nodes in the oedematous mesentery—which supports his contention that it is easy to miss evidence of previous inflammation. We did, however, observe numerous tubercles on a portion of the dilated ileum.

The strangulated bowel was clearly beyond hope of recovery, and the two loops were therefore excised in one piece, together with the defect in the mesentery, and continuity of the bowel was restored by axial anastomosis. The post-operative course was surprisingly uneventful; gastric suction was not employed, as it was felt that a tube in the pharynx might aggravate the chest condition, and in fact he did not vomit after the operation. After some hesitation intravenous fluid was begun at midnight on September 11/12, but the needle escaped from the

vein before 500 ml. had been administered, and, as bowel sounds were then audible, no further intravenous fluid was given. The bowels acted after a glycerin suppository on September 14 and naturally on the 16th. In view of the risk to other patients in the ward he was transferred back to the sanatorium the next day. His cough then appeared no worse than before his operation. On January 3, 1952, his chest condition remained unchanged and he has had no further abdominal symptoms.

Dr. M. Symons reports as follows on the specimen: "Approximately 100 cm. of small intestine, purple in colour, and containing a foul-smelling mixture of blood and faeces. At the centre there is a hole in the mesentery, through which loops of gut from above and below have passed in opposite directions. On the gut side of the hole are a number of large hard lymph nodes. Microscopically the mesentery shows extensive haemorrhage into loose connective tissue; it contains lymph nodes which are almost entirely replaced by hard tubercles with no caseation."

Comment.—Gangrene of the bowel appears to be uncommon in transmesenteric hernia, and I am not aware of a previously reported case in which the hernial orifice was available for histological examination.

I am indebted to Mr. Baty for making his paper and the record of his case available to me, to Dr. Harker for anaesthetizing this poor-risk subject, and to Dr. Symons for his careful examination of a somewhat unattractive specimen.

J. A. RUSSELL JOHNSON, M.B., Ch.B., F.R.C.S.,
Consultant Surgeon, Royal Salop Infirmary, Shrewsbury.

Spontaneous Rupture of the Stomach

Cases of this condition are extremely rare. According to Leger and Maes (1947) only 30 cases have been reported. A further case was recorded by Ladkin and Davies (1948). I have been able to find only four cases in the British literature, and all ended fatally. The present case is, I believe, the first recorded survival in the British literature and the second in the whole literature.

CASE REPORT

A married woman aged 67, the mother of ten children, was admitted on December 3, 1949, for an "acute abdomen." She had been healthy until 1939, when she began to suffer from indigestion and pain in the epigastrium, and *x*-ray examination revealed a gastric ulcer. Operation was refused, and for ten years she was treated medically. In spite of this, she suffered from intermittent epigastric discomfort, sometimes accompanied by nausea and vomiting. Her appetite remained good. Her bowel habit and micturition were normal.

Three weeks before admission the symptoms became progressively worse in spite of greater care in diet. The vomiting became frequent; it was pronounced at the end of the day, and latterly every meal had been vomited. The vomiting gave some relief from the discomfort and fullness. Her last normal meal was three days before admission. The next day she had only a small meal, and the day before admission she spent in bed without taking any solid food. Repeatedly during the 48 hours before admission she unsuccessfully attempted to vomit; the pain in the epigastrium was more pronounced, and she was seized with sharp stabbing pain in the left hypochondrium, which lasted a few seconds and relieved the desire to vomit. This acute pain was then replaced by a constant dull pain generalized in the upper abdomen, maximal at first in the left hypochondrium and later in the left iliac fossa.

On examination her general condition was poor; she was shocked, dehydrated, and very apathetic. Her tongue was furred and dry. Her pulse was 112, respirations 32, and temperature 97° F. (36.1° C.). The cardiovascular and respiratory systems were normal. There was obvious wasting. The epigastrium was distended, and tenderness and rigidity were present throughout the abdomen, with the maximum point of tenderness in the left hypochondrium. A tentative diagnosis of perforated gastric ulcer was made and immediate operation carried out.

Under nitrous oxide, oxygen, and ether anaesthesia (Dr. C. R. Blaiklock) the abdomen was opened through a midline supra-umbilical incision. A small amount of turbid fluid was at once apparent. The stomach was grossly distended, the greater curvature being well below the umbilicus. The wall was thin, smooth, and greyish white in colour. The pyloric antrum was thickened, causing complete obstruction, but the duodenum appeared normal. A large amount of greenish grey, turbid, slightly offensive fluid, with fragments of undigested food (peas, onions, and raisins) was found in the left hypochondrium. On handling the stomach, some of the contents escaped through a linear slit in the anterior wall fairly close to the lesser curvature and near the cardia. The slit was 1 in. (2.5 cm.) long, running parallel to the lesser curvature. The edges were clean-cut and paper-thin, and there appeared to be no local pathological lesion. Examination of the surrounding stomach wall revealed no evidence of acute or chronic inflammation, necrosis, or previous scarring. There were no adhesions. A short distance from the slit, near the cardia, the loss of a circular-shaped portion of serosa was noticed. No evidence of any other pathological lesion was found in the region of the rupture.

Closure of the slit was not easy owing to the thinness of the edges of the stomach wall. A continuous suture was inserted, then a second Lembert layer suture, and a stomach tube was passed for lavage. Some difficulty was encountered owing to spasm of the cardia. Because of the pyloric stenosis a posterior gastro-enterostomy was performed. The soiled area was swabbed out. During this operation the capsule of the spleen was damaged, and profuse haemorrhage followed, necessitating splenectomy. The abdomen was closed and drainage was established through a separate incision in the left hypochondrium.

The patient made a satisfactory recovery and left the hospital three weeks after the operation. One year later she had gained weight, was taking ordinary diet, and was enjoying a normal life.

A radiological report on April 17, 1950, by Dr. B. Klukvin stated: "Oesophagus normal. No deformity of the stomach could be seen at the site of rupture. Meal entered the stomach freely. Posterior gastro-enterostomy functioning normally. No barium could be persuaded to pass the pylorus."

COMMENT

A study of the literature reveals that various factors must be considered as the causes of spontaneous rupture of the stomach. The distension of the stomach, with the increase in gastric pressure, is a striking feature. It may be associated with overloading or overdrinking, sometimes combined with the administration of bicarbonate of soda. It may occur with or without any pathological condition. It may follow after operation, and in one instance occurred during labour.

In the case reported pyloric obstruction was complete and associated with spasm of the cardia. No doubt this condition was accompanied by reflex dilatation of the stomach—the intragastric pressure exerted by paroxysmal aerophagia, retching, and violent, almost unproductive vomiting, steadily increasing until rupture of the thin wall relieved the pressure.

I should like to thank Mr. R. J. Rutherford for permission to record this case.

R. REJTHAR, M.D.,
Late Senior Registrar, Ashington General Hospital, Northumberland.

BIBLIOGRAPHY

Carson, J. (1846). *Edinb. med. surg. J.*, **66**, 28.
Cavaillon and Leriche, R. (1908). *Lyon méd.*, **111**, 84.
Ladkin, R. S., and Davies, J. N. P. (1948). *British Medical Journal*, **1**, 644.
Leger, L., and Maes, J. (1947). *J. Chir., Paris*, **63**, 35.
Miller, J. R. (1933). *New Engl. J. Med.*, **209**, 1085.
Steinmann, F. (1917). *Zbl. Chir.*, **44**, 180.

Reviews

ATLAS OF BONE DISORDERS

An Atlas of General Affections of the Skeleton. By Sir Thomas Fairbank, D.S.O., O.B.E., M.S., Hon.M.Ch.(Orth.), F.R.C.S. (Pp. 411; 184 illustrations. 55s. net.) Edinburgh and London: E. and S. Livingstone. 1951.

Sir Thomas Fairbank's preface to his book and its dedication might lead the uninitiated to think that he has been most fortunate in his professional associations and that his friends have taken special pains to encourage him in his work on general affections of the skeleton. This impression would be wrong. The fact is that scores of his colleagues, bewildered by the odd things that can go wrong with bones, have for years turned for help to the one man who has had the enthusiasm and the patience to delve into this bizarre field of medicine. The experience of calling on him is not easily forgotten. A house-surgeon sent round to see Sir Thomas (because his chief is too busy) is received as a colleague. The history and the radiographs are studied in a leisurely way and with an interest that makes the tyro feel he has brought something that must be at least the medical equivalent of a gold stater of Alexander the Great. Then there is the hunt through the archives for a similar case or a near likeness, a discussion of the possible diagnoses, finally the opinion, and, "I'd like a copy of that, if you don't mind." And now this connoisseur, having given us a foretaste in the British numbers of the *Journal of Bone and Joint Surgery*, has completed the task of committing his unique knowledge to paper in book form.

The classification embraces about 80 conditions, grouped more or less according to aetiology: congenital developmental errors; acquired affections of unknown origin; disorders due to errors of diet and metabolism, those due to endocrine upsets, those due to infection or toxaemia, those connected with diseases of the haemopoietic and lymphatic systems; and multiple neoplasms. The introduction is hard going, but it must be read, because it contains most of the keys to differential diagnosis; the tables of features such as dwarfism and increased density of bone are particularly valuable. The author discusses each disease in an essay, long or short according to the importance of the condition, embellished by case records and numerous illustrations, over 500 in all. Here the general reader will find all he wants to know and more, and the research worker seeking ultimate detail will usually be guided to it by the many well-chosen references. The quality of the illustrations entitling the book to be an atlas reflect great credit on the publisher, and it should be remembered that many of the films were taken at a time when radiographic technique was still very imperfect. The choice of pictures is excellent, too, but for this very reason the occasional lapses are all the more noticeable. Thus the radiographs illustrating hyperparathyroidism, which for reasons of differential diagnosis has parted company with other endocrine disorders, are uniformly poor. Osteogenesis imperfecta, a condition easy to recognize, enjoys no fewer than 43 illustrations; there are 31 for osteopetrosis. But eosinophilic granuloma, perhaps because "there is nothing characteristic and distinctive in the [radiographic] appearance . . . and the diagnosis may be difficult," is represented by only two. This is a pity.

The difficulties in the selection of material must have been great, and there are a number of targets for the destructive critic. Where should the line be drawn? Why, for example, include congenital syphilis and miss out multiple osseous tuberculosis and yaws? By contrast a freakish condition, the Laurence–Moon syndrome, puts in two appearances. And the endocrinologists might complain that there is no orderly exposition of the influence of hormones on skeletal development. It requires no special intelligence to find faults of this kind, for Fairbank has had to contend with all the problems that plague the compilers of the geographical atlas. Snapper, in his famous monograph, made no attempt to be comprehensive; he wrote unrestrainedly about the bone diseases that interested him, and ignored the rest. Casuccio's *Osteopatie Rare*, though fairly comprehensive, is not in the same class as Snapper's and Fairbank's works, because it lacks the authority derived from long personal experience. How numerous and complex are the difficulties of the atlas-maker will be known to those who have studied the new Oxford *Atlas* and have read some of the more thoughtful reviews of it. It took a battalion of experts to produce 111 pages of maps and a gazetteer, and even they have not pleased everybody, nor could they hope to do so. Here we have a medical atlas of territories still only partially surveyed, and it is the work of one man. He has succeeded in covering a most intricate subject within reasonable compass, and has produced a book so readable that it will be enjoyed by many whose interest in general disorders of the skeleton is usually remote. This is an achievement of which he has every reason to be proud. For years we have sought in vain for an atlas like this; now we have it.

When a book immediately earns a place in the front rank of medical literature, as does Fairbank's *Atlas*, it may reasonably be suggested that arrangements for further editions should be made without delay. New and better illustrative material will be needed here and there. But, far more important, the subject of general bone diseases is a lively one; knowledge is accumulating fairly rapidly, and the pattern of ideas changes. Sir Thomas Fairbank refuses to grow old; his *Atlas* must not be allowed to do so.

H. J. SEDDON.

BONE AND JOINT DISEASES

Bone and Joint Diseases: Pathology Correlated with Roentgenological and Clinical Features. By J. Vernon Luck, M.S.(Ortho.), M.D., F.A.C.S., F.I.C.S. (Pp. 614; 448 figures. £6.) Oxford: Blackwell Scientific Publications. 1950.

In this book, dedicated to Arthur Steindler, the author has been remarkably successful in relating the pathology of bone and joint diseases to the clinical and radiographic findings. There is every evidence of careful compilation from lecture notes of high teaching value. The text is clear, informed, and well balanced; the illustrations have obviously been chosen from a mass of good clinical material; and the selection of references at the end of each chapter makes interesting reading.

The only real faults in a production otherwise first-class are the indifferent quality of a number of the radiographs and the disorderly arrangement of figures in relation to text. The blockmakers have failed to reproduce the detail of a number of good original films to such an extent that the lesions have had to be indicated by arrows. In other films the significant portion (for example, one finger out of a whole hand, or three lumbar vertebrae out of a large plate embracing the hip-joints and the lower ribs) has not been selected for enlargement to a suitable size. The impact of the illustrations has been impaired time and again by their being placed away from the corresponding text. Three of the four illustrations of osteoid osteoma, for example, come before the text is in sight, and when that section does come it is trisected by two large slabs of giant-cell tumour. But these errors of make-up will prove to be nothing more than the teething troubles of the first of many editions of a splendid teaching work.

K. I. NISSEN.

MANAGEMENT OF THE NEWBORN

Management of the Newborn. By Professor A. H. Parmelle, M.D. (Pp. 358; 47 figures. $7.) Illinois, Chicago: The Yearbook Publishers, Inc. 1952.

In a thoroughly practical book Professor A. H. Parmelle describes the management of the newborn with a wealth of personal experience to guide him. He discusses just enough of the theoretical considerations to justify the procedures advocated, and his style makes for easy reading and

easy understanding. His suggestion of a "delivery room life-saving team" for hospitals with a large obstetrical department might do much to reduce the numbers of deaths still ascribed to asphyxia. References given at the end of each chapter are almost without exception to American work, so that it is particularly disappointing to find Gairdner's views on circumcision quoted with approval but without any reference to the original paper.

The book appears to be in a series of "general practice manuals," and it is an unfortunate paradox that in fact more and more newborn babies are delivered in hospital and never come under the care of the general practitioner at all. For those who still retain some midwifery work, Professor Parmelle's book can be cordially recommended.

ALAN MONCRIEFF.

BODY MOVEMENT

Human Kinetics and Analysing Body Movements. By T. McClurg Anderson, F.F.Ph. (Pp. 287; 238 figures. £1 10s.) London: William Heinemann. 1951.

The rationale of this book is a detailed study of the action and influence of individual muscles of the group concerned in any movement, and the author believes that such a study will lead to the prevention of disabilities associated with industrial strain and fatigue and to improvement and increased efficiency in athletic performances. Two criticisms appear pertinent. First, the details are too elaborate for readers who lack the advantage of a medical training. Secondly, however interesting as an academic exposition, it can have little practical value: in analysing the components of a muscular movement one recalls the impish and successful attempt of the toad to discomfit the centipede.

But criticism is modified by the evidence that the author is an experienced and essentially sensible student of athletics. While analysing the stages of progression in walking, running, jumping, swimming, and feats of strength, and embodying the details by means of line drawings made from cinematograph films, he is careful to warn that analysis must be preceded by a preliminary assessment of the general characters of movements as seen in actual performance, and that Procrustean generalizations must be avoided. He warns the athletic aspirant against slavishly copying a successful performer without considering individual requirements; he recognizes the necessity for adjustment to abnormalities, as when he indicates the many faults revealed in first-class exponents and emphasizes that the same ultimate result is possible by widely different methods. He is perpetually insistent on the necessity for "relaxation," emphasizing the correct meaning of the term and the frequent misconception when it is used to mean merely "slouching."

This book can be warmly recommended to physiotherapists and to football and athletic coaches and trainers. It is by no means easy to read, and in parts demands for its understanding considerably more anatomical and physiological knowledge than the author perhaps realizes, but it amply repays a careful study.

ADOLPHE ABRAHAMS.

U.S. PUBLIC HEALTH SERVICE

The United States Public Health Service 1798–1950. By Ralph Chester Williams, M.D. (Pp. 890; illustrated. $7.50.) Washington: Commissioned Officers Association of the U.S. Public Health Service (P.O. Box 5874, Bethesda 14, Maryland). 1951.

Dr. R. C. Williams is qualified by 37 years of experience in the public health service of the United States, which include nine years of editorial duties, to write its history. He has brought not only wide personal knowledge but enthusiasm and imagination to his task. Undaunted by the vast scope of his subject in time, space, and range of activities, he has marshalled his facts in twelve chapters. For historians and for British readers perhaps the most interesting are the first two, which are on the history of the U.S. Marine Hospital Service 1798–1902, from which the whole Federal health service evolved, and on the history of quarantine, which led to a wider concept of the control of disease. In both these chapters the spread of ideas from Europe to the United States is evident, but the rapid growth of a nation drawn from so many and diverse sources, with peculiar epidemiological and social problems and with climates ranging from the arctic to the subtropical, has resulted in measures which are wholly indigenous and from which the Old World can learn much.

The subjects range from the control of plague, yellow fever, malaria, and typhus to dental caries, in which laboratory research, field studies, and administrative measures have all played their part. In a particularly interesting chapter intra-governmental relations are discussed and the work of the Public Health Service is revealed in all its magnitude, this being the only Federal agency which deals with all international, State, and local health authorities.

Were the book confined to abstractions and generalizations it would be not only much less bulky but more familiar in style to British readers, schooled in the tradition of anonymous service. Its length is, however, increased to 890 pages by the inclusion of much personal information. There are biographies of the directors of the National Institutes of Health, while the second world war is described largely in terms of the experiences of public health officers. Their names account for much of the forty-page index and their photographs for a high proportion of the two hundred illustrations. Although the book is excellently printed, these photographs are very poor in quality and the binding is inadequate for a book which weighs over five pounds. The mingling of personal experiences of slight or transient significance with descriptions of important developments in preventive medicine, while providing a human interest, gives the book an uneven quality. Since such presentation is likely to become the standard one for works covering world medicine, it is worth considering, before condemning the almost embarrassing intrusion of the personal element, whether the total exclusion of the individual records might not have a dehumanizing effect.

J. J. KEEVIL.

MEDICAL SYMPTOMS

Major Symptoms in Clinical Medicine. By John Almeyda, M.R.C.P., D.P.H., M.R.C.S. Volume 2. With foreword by Sir Adolphe Abrahams, O.B.E., M.D.(Camb.), F.R.C.P. (Pp. 370–704; 137 illustrations, including six in colour. £1 5s.) London: Henry Kimpton. 1951.

Dr. Almeyda's second volume is on neurology, psychology, dermatology, locomotor diseases, and endocrine disorders. It must be said that it contains much the same faults as were noted in the first volume (*Journal*, August 26, 1950, p. 499). The information it contains is poorly arranged, out of perspective, and sometimes open to doubt. The presentation is often reminiscent of a type of student's answer which examiners always find annoying. A string of questions for the patient is suggested, or one is advised to pay particular attention to special points in the physical or special investigations without indicating why.

Passion is defined as an excessive desire for sexual intercourse, which is surely a misuse of the English language, and it is apparently limited to the female sex. In its investigation one is advised to pay attention, among other things, to the pineal gland. It is true that the ancients thought that the pineal was the lodging place of the soul and that pineal tumours sometimes produce precocious puberty, but neither of these observations has much to do with sexual frustration or nymphomania, which Dr. Almeyda regards as the basis of passion. Under insomnia, again, the important distinction is not made between difficulty in getting to sleep and too early waking; malingering is overemphasized and the psychoneuroses and psychoses are underemphasized. Like the first volume, the book is well produced at a reasonable price, but some of the original illustrations are poor.

L. J. WITTS.

BRITISH MEDICAL JOURNAL
LONDON
SATURDAY AUGUST 9 1952

CRITICISM OF THE WORKING PARTY'S SCHEME

A letter by Mr. Hilary Marquand in this week's *Supplement* will raise doubts among general practitioners about the distribution of the Danckwerts award. The Supplementary Estimates have been voted in the House of Commons, but there has been no debate on the Working Party's report, which has been approved both by the Conference of Representatives of Local Medical Committees and by the Representative Body at Dublin. It has also, as we pointed out in a leading article in the *Journal* of July 5, been approved by the Assistants and Young Practitioners Subcommittee. As the House of Commons rose without any debate on the Working Party's scheme, it has been widely, and we think correctly, assumed that the whole matter is finished and done with, at least until some entirely new situation arises calling for a fresh examination of the remuneration of general practitioners and the method of paying it. But in his letter Mr. Marquand, who as previous Minister of Health must be speaking with the authority of the Labour Party, says that the Opposition will seek an opportunity when Parliament meets in the autumn to make its own proposals on the distribution of the Danckwerts award. He draws attention to the statement made by the Parliamentary Labour Party after it had studied the Working Party's scheme, incidentally now accepted by the Government. The Labour Party considers that "a most valuable opportunity has been missed to better the general practitioner service to the public and to improve the standards of doctoring throughout the country. Moreover, the report fails adequately to improve the position of the young and poorer-paid doctors having small lists of patients, and gives the greatest increase of payment to those who are already receiving higher rates of remuneration. Its proposals for the encouragement of group practice are grossly inadequate." Mr. Marquand says he has had support for these criticisms by letter and by deputations from large numbers of doctors—just how many he does not say. There has been some criticism of the scheme of distribution in letters published in recent issues of the *Supplement*.

The criticisms boil down to this—that the scheme does not facilitate the entry of the younger doctor into practice, and gives little or no relief to the elderly doctor with a small list or to those, young or old, with a medium or small list. Just how valid are these criticisms? First of all the volume of them is small. Both the "panel conference" and the Representative Body, between them adequately reflecting the opinion of general practitioners in this country, have endorsed the scheme of distribution, as have representatives of assistants and younger practitioners. To ignore this powerful evidence, as Mr. Marquand and others who think likewise with him have, is to ignore the most important fact in the whole situation. One should note too that the conference accepted Dr. Wand's rider that a part of the "final settlement moneys" should be used for making good any serious defects in the distribution.

The discontented and apparently Mr. Marquand want to load the capitation fee for the first thousand of a doctor's list. Is it then being asserted that the average, or shall we say the ideal, list is one of a thousand? If this is the case, then there are certainly not enough general practitioners in Britain to look after the people in it. If one divides the number of practitioners in England and Wales into the population of this country, then an average of about 2,200 patients to each general practitioner is reached. This being so, and in view of everyone's agreement on the desirability of distributing general practitioners more evenly throughout the population, a case could be argued for loading the capitation fee for the second thousand in a doctor's list. The loading is on the thousand persons on a list after the first 500, giving a capitation fee of 27s. for numbers 501 to 1,500 on a list.

A sum of money has been set aside to meet the case of the elderly doctor, who, by the nature of things, can undertake less work than formerly. At the same time it should be remembered that the elderly doctor will have his compensation on the value of his practice and presumably during the course of his life has made provision for his latter days. Everyone, of course, sympathizes with the position of the elderly doctor, but a scheme of distribution cannot possibly be governed by the existence of a small number of persons reaching the end of their career. As for the younger doctor, his representatives have already expressed approval of the Working Party's report—a fact that needs to be repeated. The restriction imposed upon lists and agreed to by the profession and the Ministry of Health will, when it operates, unload work from the overworked practitioner and help to distribute it more evenly among those with

smaller lists. The initial practice allowance makes it easier for the young man to start work on his own in areas yet to be designated by the Medical Practices Committee. The notional division of lists makes it financially more attractive for a principal to take on a partner. Until health centres are available, which will not be for many a long day yet, group practice must be formed around partnerships, and partnerships are being encouraged.

Obviously no scheme of distribution could possibly be expected to satisfy everybody. The criticisms levelled against the scheme are no doubt sincere and reflect the fact that some general practitioners in the Health Service—but surely not a great number—are disappointed. This is inevitable. Yet the criticisms on examination seem to have peculiarly little validity, and they have been made before the scheme has had a chance to work. Some are based on the assumption that the scheme will not turn out to the greatest advantage of the majority of doctors and to the majority of the population—an assumption which by the nature of things can be only speculative and not based upon experience. The one principle that does seem to have been affected by the scheme is one which has passed almost unchallenged, and that is the principle of free choice. Further restriction of lists does sharply challenge this principle—the freedom of the patient to choose and to change his own doctor. This has always been one of the fundamental principles of the B.M.A., and, even though there has been acceptance of the view that a large list may throw too much work on the doctor, with perhaps some detriment to the patient, nevertheless a further inroad has been made into what most people regard as fundamental in the doctor–patient relationship—freedom to choose and to change.

Mr. Marquand and his colleagues in the Opposition must, one assumes, be right when they state that they can arrange for a debate in the autumn on the Working Party's scheme, and if by a freak of fortune they were to come into power then they have signified their intention to alter the method of distribution of the Danckwerts award. But short of this general practitioners can be assured that the Danckwerts award will be distributed according to the Working Party's scheme, which has been accepted by both the profession and the present Government. This being so, there would seem to be little point in the Opposition trying to stage a debate next autumn, as it will only afford an opportunity for airing views which will be heavily biased by party politics. If the Health Service is to be of real value to the community and is to be worked by a relatively satisfied medical profession, then the sooner the Health Service is taken out of party politics the better.

CLOSED BOOK

The persistent attempts by Durham County Council to impose its closed-shop policy on its medical and certain other professional employees has at last been defeated.[1] The Board of Arbitration has been as quick to dispose of Durham's pretensions as the county council has been tardy throughout the last two years in coming to its senses. Having given an undertaking before the arbitration that it would abide by the Board's decision, the county council is now expected to affirm that the disputed regulations will no longer apply to the professional people concerned. These are the doctors, dentists, nurses, midwives, teachers, and professional engineers.

The dispute itself was over an issue that some might regard as trivial: the regulations required an employee to apply for extended sick leave with pay through his professional organization or trade union. As a consequence an employee not a member of such an organization was deprived of any right to make this application, and the regulations therefore indirectly compelled him to join one if he wished to enjoy the same advantages as his colleagues who were members of a professional organization. But though this peculiarly oblique form of the closed-shop policy may appear trivial at first, it has to be seen in the light of Durham's long-continued attacks on what professional opinion rightly regards as a cherished freedom of professional men and women. For nearly a year during 1950–1 the county council was requiring evidence from its medical employees that they belonged to a professional organization or trade union, and threatened to dismiss any doctors who refused to give the evidence. The doctors, loyally backing the policy of the B.M.A., refused to comply under this threat, and in the upshot none were dismissed. In June last year Durham advertised certain medical vacancies in the lay press, and these were made the subject of an "Important Notice" in this *Journal*. The B.M.A. informed the Ministers of Health and Education of what was going on, but the Ministers decided not to intervene. A few weeks later, no doubt as a result of the "Important Notice" procedure, Durham capitulated and agreed not to ask doctors about their membership of professional organizations. Meanwhile it had passed the regulations recently in dispute, and adamantly contended that they were not a part of the closed-shop policy. No further progress could be made against the stubbornly held opinions of many of Durham's councillors, though the Joint Emergency Committee of the Professions, formed in March this year, found a compromise that an emergency committee in Durham

[1] See *Supplement*, p. 91.
[2] *The Times*, July 31.

was willing to accept. Unfortunately the county council did not attach the importance to its own committee's decisions that might have been expected, turned down the compromise, and ultimately, when wiser counsels prevailed, agreed to go to arbitration.

This unhappy episode is now over, and normal relations should again be established between the county council and its professional employees. The chairman of the county council is reported to have said[2] that the findings of the Board "are a great disappointment to us." No doubt they are, but it is to be hoped that the disappointment will not be allowed to rankle and so to damage those harmonious relations between employer and employee without which no service can be satisfactorily run. The B.M.A.'s policy on the closed shop has always been clear without equivocation: while welcoming and encouraging doctors to become members of the Association of their free choice, it will not allow them to be coerced into membership. This policy not only commands entire assent throughout the professions but has in fact been accepted by the United Nations, of which Britain is a member. The Universal Declaration of Human Rights, adopted by the United Nations General Assembly in 1948, has as its Article 20 (2): "No one may be compelled to belong to an association." It is the more to be regretted that the professions seem to be almost alone in Britain in standing up for this elementary right of human beings, and this the first success of the Joint Emergency Committee of the Professions suggests that much can be attained when the professions combine to protect their freedom and traditions. Much credit is due to the Committee for defending so fruitfully the liberties of professional men and women in Durham, nor should it be grudged to those in power at Durham who had the wisdom to agree to refer the issue to independent judgment.

PROBLEM OF THE FEEBLE-MINDED

Last week Dr. J. F. MacMahon (on p. 254) called attention to the difficulty of providing adequate care and treatment for the mental defective. Under the present system of control of hospital and community care defectives are split into several categories for which different authorities are responsible, and the provision of beds in mental deficiency institutions and of places in special schools for educationally subnormal children is inadequate: 28,000 children are competing for 15,000 places. Ideas on mental deficiency are changing, and the law has not kept pace with this evolution.

Practice, however, has changed greatly since 1913, when the first Mental Deficiency Act was passed. At that time children who needed institutional care were not regarded as likely ever to make sufficient recovery to justify discharge to their homes or to free community care. The new definitions adopted in the Mental Deficiency Act, 1927, were intended by Parliament to include in particular those under the age of 18 years who had suffered from the ravaging effects of the epidemics of encephalitis lethargica of 1921 and 1924. In the discussion which took place during the passing of the Bill the needs of epileptics who might deteriorate during adolescence (before the age of 18 years) were also put forward as urgent. It would seem from this that the definitions were intended to cover not merely intelligence but that "defect of mind," the partial failure of the wider mental functions, which results in social inefficiency. As Dr. MacMahon remarks, it seems that there are now difficulties in including the high-grade defective patient within the terms of the 1927 definition. Of the 3,000-odd admissions each year to mental deficiency institutions the proportion of low-grade patients (those with an I.Q. of 49 or under) has increased and continues to rise. Fewer anti-social subnormals and high-grade defectives are coming into institutions than in former years. The high-grade group are either being dealt with according to the processes of law in the courts and are no longer regarded as "defective," or in a period of full employment have a value in the labour market which enables them to be self-supporting.

Dr. MacMahon, in suggesting separate legislative provision for "high-grade aments," whom he defines as low-grade normals, is reflecting the widely accepted view that there is a borderline group of inadequate or socially aggressive individuals who are neither wholly normal, nor defective, nor even mentally ill. They are the odd men out, the inadequates for whom we want to do so much and for whom we have in the past been able to do so little. They cannot all be dealt with under the Mental Deficiency Acts, and indeed such a course would be undesirable and perhaps legally impossible. But, if new legislation is needed, none can say just how these low-grade normals should be compelled to accept control and training without restricting their liberty for the period that treatment demands. We may discard words like "idiot" and "feeble-minded," which are socially offensive and hurtful to the family, but it does not appear that we have yet coined terms which will express social aggression or inadequacy clearly enough to justify putting into the hands of doctors and magistrates the powers to enforce treatment under strictly controlled conditions.

The *International Classification of Diseases*, published by W.H.O., defines an imbecile as a child having an I.Q. of 20–49 or an adult with a mental age of

3–7 years, and a feeble-minded person as one with an I.Q. of 50–69 or a mental age of 8–12 years. Dr. MacMahon suggests that high- and low-grade defectives (the feeble-minded, and the imbecile and idiot) should be treated in separate institutions. The Hedley Report of 1931 and the Cost of Hospitals Report of 1938 support strongly the idea of mixed institutions for both sexes and all grades. The broad generalization of mental deficiency practice suggests that the most economical and most practical arrangement is one in which the grades are classified into groups separately housed but loosely associated in one large institution of not fewer than 880 beds. But for high-grade patients (mental age 10 years and over) we recognize the need for concentrated study, and prolonged training and treatment, in the effort to produce social recovery and the resumption of ordinary life in the community. It is possible that at least some of these patients could be selected for treatment with others who are suffering from social inefficiency but are not true defectives. Both groups need prolonged training, supplemented by treatment from experienced psychotherapists. The problem needs further thought and inquiry, but at least we are recognizing the need and are facing the problem of finding a solution which does no true violence to the individual.

TREATMENT OF LEPROSY

The detailed diagnosis of leprosy and the classification of the type are important in determining the best line of treatment. The leprologist is therefore depending more and more on the lepromin test and the study of the histopathology of the lesions of the skin. In doubtful cases it is wise to send a biopsy specimen from the suspected lesion, including if possible a small subcutaneous nerve supplying the area, for detailed examination. Sections should preferably be fixed in Zencker's solution, washed for 12–18 hours, and dispatched in 40% spirit: formol saline may be used for fixation but is less satisfactory. Work is being done in South America, and by French observers, on the effect of B.C.G. vaccination on the lepromin test. There is evidence that a negative lepromin reaction can be converted into a positive by vaccination, and the results of further investigation will be awaited with interest.

Sulphone therapy has now become the standard treatment of the disease. For cases seen in this country 25 mg. of diamino-diphenyl sulphone (D.D.S. or "dapsone") can be given as a commencing dose twice a week. This should then be increased by 25 mg. every 14 days until 100 mg. twice a week is reached. The dose is then further increased by 100 mg. each month to a maximum of 400 mg. twice a week. Alternatively sulphetrone in aqueous solution (50%) can be given by deep subcutaneous or intramuscular injection, 0.5 ml. twice a week. This dose should be increased by 0.5 ml. each month until 3 ml. is given twice a week. Thiosemicarbazone is generally believed to be less effective than the sulphones, but it is a useful stand-by for patients who show intolerance to the sulphone group of drugs. The commencing dose is 25 mg. daily, increasing by 25 mg. every week until 150 mg. per day is given.[1 2] Lepra reactions necessitating cessation of sulphone treatment may occur, and their treatment has been described by Cochrane.[3] Specific therapy at half the dose which produced the reaction should not be recommenced until all signs of reaction have subsided. Children of 12 and under should receive half the adult dose of the drugs. Sulphone therapy should be continued for at least one year—and in this country two years is advised—after standard methods of examination have yielded negative results, or, if the case is non-lepromatous, for 18 months after all activity of the lesions has ceased.

In most institutions abroad sulphone therapy has very largely replaced hydnocarpus treatment. The general tendency, because of its ease of administration and cheapness, is to use oral diaminodiphenyl-sulphone ("dapsone") in twice weekly dosages. The commencing dose is usually 100 mg. twice a week, increasing by 100 mg. each month to 200–300 mg. twice a week. Where adequate supervision is not possible it may be preferable to inject aqueous sulphetrone in a 50% solution subcutaneously. The commencing dose is ½–1 ml. twice a week, increasing by 1-ml. increments every month until 3 ml. twice a week is given.

Recently cortisone has been advised for lepromatous reaction, particularly in that type known as erythema nodosum leprosum. But it has recently been found that, while cortisone temporarily stops the reaction, it later results—as one might expect—in the spread of the disease. For severe nerve pains 4 ml. of a 2% solution of procaine injected into the nerve sheath will be found useful. If the nerve is swollen and tender, particularly in tuberculoid cases, the nerve sheath should be excised surgically.[4] The necessity for physiotherapy and orthopaedic measures, including operative procedures for the relief and prevention of deformity, must be borne in mind, so that the patient, on recovery from leprosy, may not become seriously handicapped through permanent contractures.

[1] Cochrane, R. G., *Practitioner*, 1951, **166**, 380.
[2] Lowe, J., *Lancet*, 1952, **1**, 436.
[3] *Practical Textbook of Leprosy*, 1947, London, pp. 136–144.
[4] Ibid., p. 146.

PROGRESS IN MALARIA CONTROL

When the insecticidal value of D.D.T. was discovered it was expected that there would be far-reaching results from its use, and that a new era in malaria control would be introduced. These anticipations are now materializing, and in British Guiana and in Mauritius important schemes have been undertaken. The results of the latter are described in a paper on page 309 of this issue; those of the former were communicated to the Royal Society of Tropical Medicine and Hygiene by Dr. G. Giglioli at a meeting on June 19 (see page 347). The ultimate effect of these and similar schemes is difficult to visualize, but they may well

profoundly alter the health, well-being, and economy of populations living in malarious areas.

Describing the results of the scheme in Mauritius, Dr. M. A. C. Dowling reports that the malaria notifications fell from 29,938 during the first six months of 1948 to 852 during the similar period of 1952, while the total death rate per 1,000 of the population fell from a mean of 27.2 for the years 1934–50 to 13.9 in 1950. The mean infant mortality per 1,000 live births for the same period was 150.0, and fell to 76.4 in 1950. It appears certain that the improvement in all these figures was the result of malaria eradication.

It is interesting and important to note that though spraying with D.D.T. alone reduced malaria and its chief vector in Mauritius—*Anopheles funestus*—to a very low level, it was not so successful against the second vector there—namely, *A. gambiae*. Accordingly a larvicidal scheme was undertaken whereby breeding of this mosquito was reduced by more than 97%. Although much has been achieved in Mauritius, few will disagree with Dr. Dowling's statement that "it would be rash to relax vigilance for many years to come."

The cost of these schemes is considerable, amounting annually to 3s. per head in Mauritius, or a total of approximately £75,000. It is possible that with chemoprophylaxis with one of the newer antimalarials similar results might be brought about more cheaply, provided the population at risk were willing and able to co-operate. This latter, however, is a variable factor difficult to control. These and many other considerations have to be taken into account in deciding on a scheme suitable for any particular region. The Colonial Insecticides Committee are to be complimented on having fostered research on these important matters and thereby put themselves in the position of valuable advisers.

Not the least of the problems involved in these schemes is their effect on food requirements of a country or region, for the population will probably rapidly increase after their introduction. Agricultural, antimalarial, and other schemes should therefore be closely co-ordinated. Fortunately, by increasing the health of a population their working capacity and therefore their food production often automatically increase. Research on the inter-relationship of parasitic diseases and nutrition is therefore important, and it is encouraging to know that at the Human Nutrition Research Unit of the Medical Research Council in the Gambia work along these lines has been vigorously and successfully prosecuted for the past six years.

RADIOGRAPHY OF THE LUNGS

In March, 1950, a committee was appointed by the Joint Tuberculosis Council, the Faculty of Radiologists, and the Society of Thoracic Surgeons to consider the terms used in reporting on radiographs of the lungs, and also to bring up to date the report on "Standards of Technique in Chest Radiography" adopted by the Joint Tuberculosis Council in 1948. The report[1] of this committee has been issued by the Ministry of Health. The first part is concerned with the terms used in reports on radiographs of the lungs, and is of interest to general physicians as well as to radiologists and those specializing mainly in pulmonary disease. Authoritative support is given to the use of the word radiograph for the *x*-ray picture itself. It is recommended that the radiologist's report should be primarily descriptive with an added interpretation, and the committee therefore defines both descriptive and interpretative terms. In describing abnormal shadows seen in a radiograph ordinary adjectives should be used to describe their number, size, shape, density, and margins. The special descriptive terms "mottling" and "miliary mottling" are defined: the first as "multiple discrete or semi-confluent shadows, generally less than 5 mm. in diameter," and the second as "numerous discrete, well-defined shadows not exceeding 2 mm. in diameter." The term "reticulation" for linear shadows forming a network is also recognized. "Infiltration" is not defined, but is included under interpretative words, with the recommendation that it should be accompanied by an expression of opinion about its cause. "Fibrosis" should be used only when there is evidence of traction on other structures. The clear statement about the usage of the words "collapse" and "atelectasis" should save much confusion: the first is used to mean diminution in volume of a segment, lobe, or lung, and the second to mean airlessness associated with diminution in volume. The report states once more the obvious but not always recognized fact that it is impossible to define the exact anatomical location of pulmonary lesions without lateral views and often other examinations, such as tomograms and bronchograms. In reports on postero-anterior or antero-posterior radiographs the lung field may be considered in zones: the upper zone is that area above a straight line running through the lower borders of the anterior ends of the second ribs; the middle zone is the area between this line and one running through the lower borders of the anterior ends of the fourth ribs; and the lower zone is the remainder of the lung field. The nomenclature of the main bronchi and pulmonary segments, approved by the Thoracic Society,[2] is described and illustrated with diagrams.

It is very much to be hoped that this attempt to standardize the usage of the terms of radiological reporting as applied to the lungs will be widely followed, but clearly it is necessary that the quality of the radiographs themselves should vary as little as possible. This is necessary not only to secure a radiograph capable of showing abnormalities in the lung but also to allow comparison of radiographs of the same patient made over a period of time and in several clinics and hospitals.

We announce with deep regret the tragic death on August 5 of Sir Jack Drummond, D.Sc., F.R.S., director of research to Boots Pure Drug Company and formerly professor of biochemistry, University College, London, and scientific adviser to the Ministry of Food during the war.

[1] *Standardisation of Radiological Terminology in Pulmonary Disease and Standardisation of Technique in Chest Radiography*, 1952, H.M.S.O., London.
[2] *Thorax*, 1950, **5**, 222.

REFRESHER COURSE FOR GENERAL PRACTITIONERS

ANAESTHETIC EXPLOSIONS

BY

C. F. HADFIELD, M.B.E., M.D., D.A., F.F.A.R.C.S.

Consulting Anaesthetist, St. Bartholomew's Hospital

For many years the danger of ignition and explosion of anaesthetic vapours has been recognized in countries like the United States, where the humidity of the air is apt to be greatly reduced by high atmospheric temperatures. It is perhaps not so generally appreciated that the same effect may be produced by very cold conditions which, owing to the freezing and precipitation of moisture, also make the air very dry. As neither of these conditions is common in this country, the danger is correspondingly less. However, during the past twenty years or so, in spite of no observable improvement in the British climate, anaesthetic explosions have become more frequent.

There seems to be no single and, indeed, no entirely satisfactory explanation why this should have happened. Hospital architects have placed sterilizing rooms at a distance from the actual operating area and so possibly reduced the humidity there by preventing the entry of steam. Air-conditioning may have reduced the humidity of the air to a dangerous degree. Possibly a leading factor may be found in the increasing use by surgeons of cautery, diathermy, and endoscopes of various kinds.

Explosive Properties of Anaesthetics

It is unnecessary to describe in detail the explosive properties of the various gases and vapours used for anaesthesia. Ethyl chloride is inflammable and possibly explosive in certain circumstances, but, probably owing to the methods by which it is used, there seems to be no record of a fire or explosion being caused by it. Chloroform vapour is free from risk of explosion, but it is well to remember that in a flame it will decompose into products which are highly irritating to the respiratory system.

The really dangerous agents are ethers of all kinds, cyclopropane, and ethylene. Explosions due to any of these are violent enough to be lethal, and therefore slight differences of degree are of little practical importance. Ethylene, which incidentally detonates more violently than the others, is now so infrequently used as to call for little further comment. However, should it be employed it may be well to remember that it is a very light gas and that consequently any source of ignition placed high enough to escape risk from heavy ether vapour might prove dangerous.

With nitrous oxide the story is very different. It is so generally and correctly regarded as an inert chemical compound that one is surprised to find that it has dangers of its own. It was the late Professor H. B. Dixon who pointed out that nitrous oxide, although non-ignitable itself, will under suitable conditions greatly enhance the ignitability and explosibility of other gases. In the special apparatus which he devised for the investigation of this and other similar problems he found that a jet of, say, hydrogen burning in air could be increased in size and heat by replacing the air by oxygen. But he found to his surprise that the flame could be still further increased by partly replacing the oxygen by nitrous oxide. The explanation is said to be that the heat generated by the flame is sufficient to split up the nitrous oxide into nitrogen and oxygen ions, and that these support combustion better than oxygen itself. Whether this explanation is correct or not, the practical lesson to be learnt from the experiment is that a mixture of, say, ether vapour with oxygen and nitrous oxide is more ignitable and explosive than one with air or oxygen alone.

The degree of ignitability and explosibility of ether and other ignitable gases varies with the strength of the vapour. A very weak mixture with air or oxygen will not ignite. Pure ether vapour will not ignite. For a similar reason it is said to be safe to drop a lighted match into the petrol tank of a car, but on the one occasion that I have heard of when this was tried the experimenter was killed. Varying mixtures of ether vapour with air or oxygen will therefore, according to the proportions, refuse to ignite, burn quietly or with a "cold flame," go off with a pop, or explode violently. So, in theory at least, if we could be sure of using a vapour either too weak or too strong to ignite, all danger would be averted. However, in practice this is quite impossible, especially with a strong vapour, as this might easily become diluted to a dangerous degree by admixture with surrounding air. The same general considerations apply to vinyl and other ethers and to cyclopropane.

None of these gases will ignite spontaneously, so their use is quite safe in the absence of any spark or flame or of any body hot enough to cause ignition. But it must be realized that a piece of metal which is not red hot, even at a temperature as much as 200–300° C. below visible red heat is still hot enough to ignite a vapour of suitable strength. As an example may be given the rubber and metal syringes sometimes used by dentists for drying cavities with hot air.

Sources of Sparks and Flames

The various sources of flames and sparks for which one must be ever on the watch may for convenience be divided into two groups—the "dynamic" and the "static." In the dynamic group can be included any source of ignition which is actually in existence or is due to the breakdown of some piece of apparatus. For instance, a coal or gas fire or an open electric stove provides such obvious sources that no discussion of them is required. Other sources of this kind are due to the breakage of electric lamps, either those connected with the main installation, or the small ones used in head-lamps, endoscopes, and similar surgical appliances. In some of these the current is reduced to the necessary low voltage by the use of a variable resistance. During his examination the surgeon often demands more light, and the adjusting button gets pushed further and further until much more than the correct two or three volts are passing to the lamp. This may cause only an inconvenient but harmless burning out of the lamp, but it may result in a fracture of the lamp or a failure in the insulation of the flexible lead; either may cause the explosion of any dangerous vapour present. In general, the use of the main electric installation, with a resistance, for endoscopic examinations is to be discouraged. It is safer to employ a low-voltage accumulator or dry battery with which dangerous overloading is impossible. Even if the correct voltage is not exceeded, there is some risk with these delicate lamps. The current is usually led to the bulb through a thin flexible insulated wire, commonly composed of a number of fine metal strands. In the course of ordinary use, such leads are frequently bent and restraightened, and it may be that first one or two and then more of the

constituent strands get broken until only a few remain intact. The danger arises from the fact that the lamp may continue to function, if not normally at least adequately. This would mean that the full or only slightly reduced current is passing through, let us suppose, not the full 20 strands of the flexible wire but a much smaller number. This must involve increased resistance, and so increased heat, which in turn may be sufficient to destroy the insulation and lead to disaster. Such a possibility can best be avoided by frequent examination and, if necessary, replacement of the flexible wire.

There are, of course, innumerable other ways in which explosive vapours may be ignited. If people will insist on investigating escapes of coal-gas with a lighted match it seems useless to exhort them. Similarly, if an anaesthetist will administer ignitable vapour near a possible source of flame he is asking for trouble. The pity of it is that his fault will involve others, and more particularly the patient. It may seem unnecessary to stress such an obvious point, but experience has demonstrated that it cannot be emphasized too often. In the Ministry of Health's memorandum entitled "Precautions Against Anaesthetic Explosions in Operating Theatres" (see *British Medical Journal*, 1951, 1, 1324) one of the accidents reported is a case in point. It reads as follows:

"A diathermy operation was in progress for carcinoma of the epiglottis. Anaesthesia was induced by chloroform (intratracheal), but during the course of the operation the anaesthetist thought it desirable to change to ether. An explosion occurred when the diathermy electrode was within the patient's mouth. Some of those present were injured by flying glass, and the patient subsequently died."

Electric switches of all kinds are specially dangerous. The ordinary electric-light switch often emits a spark, as may be confirmed by removing the cover and operating one in the dark. All such switches should be outside the theatre or, if this is impossible, should be placed as high up as may be, so as to be out of reach of any ether vapour that may be accumulating along the floor. A more serious danger is associated with the foot switches used by surgeons to control diathermy apparatus, electric saws, and the like. If operated by the foot these are of necessity on the floor, and so in the most dangerous position possible. Such switches, and so far as possible all switches near the patient, should be of the mercury "non-sparking" type in which the make and break occur in a closed tube. Electrically operated suction pumps are another source of danger. An electric motor may emit sparks, and if, as in many of the older forms of pump, the mixture of blood and explosive anaesthetic vapour is drawn directly into it an accident is almost sure to happen. Even with the safer modern types the electric motor should be outside the theatre.

Static Electricity

Explosions due to static electricity are very different from those due to exposed flames or electrical failures. Such accidents are often so unexpected that it is difficult, but nevertheless necessary, to be continuously on guard against them. When two bodies differing in electrical potential are brought into close proximity with each other a spark is apt to jump across the intervening gap. Instances of such static sparks are common, such as the slight ones obtained in frosty weather when we comb our hair or stroke the domestic cat. In some dry climates it is possible, and I believe customary, to light the gas by shuffling along the carpet, so charging oneself with electricity, and then approximating a knuckle to the metal of the gas jet. The resulting spark will light the gas just as well as that from the flint in a modern gas fire. It is in fact of great importance to realize the ease with which persons and things can become electrically charged if the circumstances are favourable. It may only be necessary to rub a cloth lightly over the rubber covering of a rubber-tyred theatre trolley to charge it sufficiently to give a spark upwards of ½ in. (1.3 cm.) in length.

The usual type of rubber-tyred anaesthetic table may become similarly charged, though it is usually very difficult to discover where the charge has come from. A possible cause may be suggested by consideration of the unpleasant experiences sometimes suffered by drivers of light steam-traction engines with rubber tyres. On a warm dry day the driver may stop for his lunch, leaving the engine standing idle. As the pressure of the unused steam rises in the stationary vehicle the safety valve will come into operation and a flow of steam escape through it at high pressure. On returning to his cab the driver may receive a shock sufficient to throw him into the road. The blast of the steam can generate a static charge which, being unable to escape because of the rubber tyres, may attain a sufficient potential to cause a disturbing shock when earthed by the driver's foot; the fitting of a trailing chain easily disposes of this trouble. The same plan is adopted for anaesthetic tables. For this purpose a looped chain is found to be more reliable than a single one, as it is less likely to be tucked away by a tidy-minded but uninstructed nurse. On the analogy of the steam-engine it seems possible that an anaesthetic table might receive a charge from the passage of gases through its metal and rubber tubes, especially should any dust be present. Experts at the National Physical Laboratory, however, have passed gases through a Boyle machine under working conditions for long periods without producing a static charge.

Some explosions seem to have been due to the charging of the anaesthetists themselves or of other persons in the theatre. Just as one may get a sufficient charge to light a gas-jet by shuffling across the floor, so an anaesthetist may by his own necessary movements charge himself to a dangerous potential if, as is usually the case, he is not earthed. In one case that was reported to me the lady anaesthetist was wearing a silk garment, and this may have been contributory. The fact that theatre personnel are generally required to wear rubber footwear naturally increased this danger, especially on a non-conducting floor. In munition factories it used to be obligatory for workers to wear rubber or felt shoes to avoid the risk of striking a spark on the floor. Such a precaution increased rather than diminished the danger. Apart from persons themselves becoming electrically charged, small pieces of apparatus, such as facepieces, metal unions, bags, and the like, may also become statically charged or perhaps obtain a charge by being handled by the anaesthetist. I know of several cases where a flame has appeared, with or without an explosion, when some metal union on the apparatus has been separated or reassembled. So real is this danger that, if such a manipulation is necessary, it is wise to discontinue the flow of ignitable gas until it is completed.

Many of the dangers from static sparks are due to the non-conductibility of the rubber of which much anaesthetic apparatus is composed. It is this that makes it so difficult for any electric potential, however generated, to leak away. During the last war one of the rubber companies succeeded in producing a conducting rubber; the general use of this for anaesthetic apparatus, wheels of trolleys, and soles of footwear would eliminate many sources of danger. Even with ordinary rubber the risk can be somewhat lessened by running a little water through the bags and tubes before use and so slightly increasing their conductivity.

Other Hazards

A special warning must be given about the handling of the ether bottle on any kind of Boyle machine. After using ether it is not enough just to turn off the ether tap before exposing the patient to any possible source of ignition. Such a tap may be entirely gas-proof, but it is difficult to be sure of this, and to be quite secure the ether bottle should be removed to a safe distance. Should it be necessary, for any reason, to leave the ether bottle in position, it is not enough to empty and replace it. This may be more dangerous than simply turning off the tap. The

"empty" bottle will probably retain a few drops of ether. A small quantity like this is far more easily vaporized than 8–10 oz. (0.25 litre) of liquid ether, and in consequence may set up a greater vapour pressure than the latter, with the attendant greater risk of leakage through a faulty tap. If the bottle is required in position it should be emptied and rinsed out with water before replacement.

Two other hazards deserve brief mention. The first concerns oxygen cylinders. It is, or should be, well known that it is dangerous to apply oil to the valves of cylinders containing compressed gases, particularly to those of oxygen cylinders. In anaesthetic apparatus the oxygen usually comes through two valves: one, the main one, on the cylinder head, and the second, a fine-adjustment valve, which controls the actual flow. If, when this second valve is closed, the main valve is *suddenly* turned on fully, the rush of released gas impinging on the second valve may generate enough heat to detonate the vulcanite of the main valve, with disastrous consequences. The presence of dust and oil will render this all the more probable. It should not be difficult to remember that the main valve (often very stiff) of an oxygen cylinder should be turned on quite gradually, and that any second valve beyond should be at least partially opened. Once the gas is flowing the valves may be operated at will without risk.

The second hazard is unconnected with anaesthesia except in so far as the anaesthetist is well situated to hold a watching brief. Surgeons sometimes make use of strong spirit lotions or actual spirit for skin sterilization and other purposes. If this is freely applied any excess may be absorbed by surrounding towels, which thus become highly inflammable. Should diathermy or other cautery be later used in the vicinity a conflagration may result.

Conclusions

When records of the actual damage done by anaesthetic explosions are examined it will be observed how variable are the resulting injuries. They are reminiscent of the vagaries of bomb damage. While many, and probably most, of these explosions have caused the immediate or delayed death of the patient, in others he has escaped unharmed. Such escapes are rare. It would almost certainly be correct to say that such an escape will never occur if the explosive gas is being administered through an endotracheal tube. In such a case there must be an open passage filled with explosible vapour extending from the site of the explosion to the trachea and bronchi, and the flash must pass along it. In this way the lung tissue will be disintegrated by the explosion or so burnt that fatal bronchopneumonia must follow.

Recent developments in anaesthetic technique make it easier than formerly to avoid risk of explosion. Non-ignitable trichlorethylene ("trilene") is of the greatest service, as it will maintain an adequate plane of anaesthesia during a dangerous period. The use of local, regional, spinal, and intravenous methods needs no more than mention. Perhaps the greatest help of all is to be found in the employment of curare and other similar muscle relaxants. With their help a satisfactory degree of muscular relaxation can be obtained without the use, or with only very limited use, of the explosible vapours.

However, in spite of all these advances, and in spite of the frequent warnings that have been published, these distressing and fatal accidents continue to occur. One short sentence sums up all that has been or can be said on this subject—namely, Never administer an ignitable anaesthetic unless the absence of any flame, spark, or other source of ignition can be assured.

Next Refresher Course Article.—"The Surgical Relief of Pain," by Professor Lambert Rogers.

CHIEF CHARACTERISTICS OF NERVE GAS

NOTES ON FIRST-AID TREATMENT

(This article has been prepared at the request of the Chemical Defence Advisory Board, and is here published by permission of the Chief Scientist, Ministry of Supply.)

"Nerve gases" are liquids with boiling points varying from 150 to 250° C. (approximately). They are therefore roughly comparable in volatility to kerosene. They are fairly soluble in water, and are slowly destroyed by hydrolysis. Alkalis, such as solutions of caustic soda or washing-soda, will decompose them more quickly. They are almost odourless and the vapour is invisible. They have no immediate irritant properties either to the eyes, skin, or respiratory tract. No warning of their presence is therefore given to the subject exposed to them.

Mode of Action

Their very dangerous and insidious qualities are primarily due to the fact that their action is that of cholinesterase inhibition—that is, they prevent the action of the enzyme which, under normal conditions, continuously destroys acetylcholine in the body as it is produced. This leads to the accumulation of acetylcholine, and hence all neuromuscular mechanisms are upset. Voluntary muscle, including the muscles of respiration, ultimately becomes paralysed and smooth muscle passes into a state of spasm. In effect there is a generalized parasympathetic stimulant action.

The total amount of cholinesterase in the body is small, and nerve gases inhibit this enzyme for a prolonged period. Therefore these gases are highly toxic and may be lethal in minute concentrations. They are rapidly absorbed by inhalation of the vapour, absorption of the liquid through the skin and mucous membrane, or by the ingestion of contaminated food or water. A drop of liquid in the eye or the inhalation of a high concentration of vapour for a few seconds may cause incapacity or death within a few minutes. Liquid on the bare skin penetrates somewhat less rapidly, but two or three drops may kill within half an hour. Ordinary clothing cannot be relied on for protection, as it is quickly penetrated.

The inhibition of cholinesterase activity may persist for many weeks, so that repeated exposures to very low concentrations may have a cumulative effect.

Signs and Symptoms of Poisoning

These can be divided into primary and secondary.

Primary, Due to Direct Action of the Nerve Gas.—There is copious salivation, slowing of the heart, constriction of the pupils, headache, nausea, diarrhoea, vomiting, muscular weakness, and sometimes generalized muscular fibrillation. The muscles of respiration are weakened or paralysed, the diaphragm being affected most. The bronchial musculature may be in spasm.

Secondary, Due to the Anoxia Brought About by Respiratory Paralysis.—This will produce cyanosis, venous congestion, low blood pressure, convulsions, and possibly pulmonary oedema. Death occurs suddenly from the effects of the anoxia.

Clinical Features.—Since comparatively few cases of poisoning have occurred, the clinical picture is as yet imperfectly known. The following summarizes the features which have been recognized.

A severe case of nerve-gas poisoning will present itself as one of acute asphyxia. The subject may be unconscious from anoxia, with cyanosis, sweating, and pallor. Breathing will be laboured and wheezing in character, similar to that of an acute asthmatic attack. While conscious, the subject will be anxious and suffering from air-hunger; there may be restlessness and lack of co-operation owing to the effects of the anoxia.

In mild cases the main respiratory symptom will be tightness of the chest and throat. There will be intense headache, constriction of the pupils, and difficulty of near vision. There may be nausea, vomiting, dizziness, abdominal pain, and diarrhoea. These symptoms may commence several hours after exposure and may persist for days if untreated.

At post-mortem examination no specific changes other than those due to anoxia are found. The accumulation of acetylcholine causes death by an interference with function and not by destruction of tissue. There is cyanosis of the viscera and blood, and congestion of the brain and conjunctivae. There may be a dilatation of the heart. The speed of post-mortem autolysis will be increased.

Treatment

To be of use this must be immediate, because of the danger from anoxia. Atropine is the antidote to the nerve gases. If there is severe respiratory distress and cyanosis artificial respiration must be started at once and maintained until adequate respiration returns of its own accord.

ANTIDOTE

Subjects suffering from nerve-gas poisoning tolerate large doses of atropine. An initial dose of 2 mg. (1/32 gr.) should be given intramuscularly. This dose should then be repeated—by either the intravenous or intramuscular route—until such time as the symptoms of poisoning disappear. A useful sign of the degree of atropinization achieved is the pulse rate, and the dose of atropine should be repeated so as to maintain a pulse rate of 110–120 beats per minute. (The signs of atropine poisoning are largely psychotic, with mania, extreme excitement, and restlessness. The latter must not be confused with the restlessness that occurs following anoxia. As much as 12 mg. of atropine sulphate in 16 hours has been given in nerve-gas intoxication.)

ARTIFICIAL RESPIRATION

The method recommended is a small hand-bellows which will ventilate the lungs in spite of the bronchial constriction. When using the hand-bellows the patient should be supine and the attendant should kneel to the right of the head (see Fig. 1). His left hand should encircle the face and hold the face-piece firmly in position; the face-piece must fit under the chin (Fig. 2). The attendant's other hand should work the bellows so that the main force is exerted at an angle of 30 degrees to the horizontal (Fig. 3). This is to ensure that the lower jaw and tongue do not fall back and obstruct the air passages. The pressure used should not exceed 20 cm. of water, since with higher pressures the lung and stomach may be overinflated. The resuscitator illustrated has a safety valve on the face-piece which blows off at 20 cm. Ventilation should be carried out at a rate of 20 to 30 times a minute and should be continued until the subject is capable of breathing adequately himself. This will probably occur within 30 minutes, but it must be remembered that respiration may fail again owing to excessive elimination of carbon dioxide during the artificial ventilatory period; hence the subject should be kept under observation. During artificial ventilation the stomach will be inflated with air by the resuscitator. This will cause some respiratory embarrassment when normal respiration is re-established. It can be minimized, however, by firm pressure over the stomach, applied intermittently to force the air up the oesophagus. During this manœuvre the patient's head and shoulders should be raised, so as to ensure that it is air and not the liquid contents of the stomach which is forced into the oesophagus. While artificial ventilation is being carried out there will be excessive salivation, and if practicable this should be allowed to drain away by periodically tilting the subject head down. If no form of positive pressure ventilatory apparatus is available, then manual artificial respiration should be performed. Oxygen, if available, should be used in conjunction with this.

FIG. 1

FIG. 2

FIG. 3

In mild cases of nerve-gas poisoning the eye symptom and headache are best treated by the instillation of atropine drops or ointment in the eye. This should be used as an adjunct to parenteral atropine therapy if necessary.

Decontamination

In the event of toxic liquid being splashed on the skin it should be washed off immediately with plenty of water. As soon as this has been done ordinary anti-gas ointment should be applied, as the subject will be unable, at this stage, to know whether the liquid is nerve or mustard gas. If the clothing is contaminated the outer clothing should be removed at once and the skin washed as soon as possible.

This first-aid treatment should be within the competence of trained non-medical personnel and, in certain emergencies, will have to be carried out by them to save life.

Reports of Societies

A NEW ANTIMALARIAL

With Sir NEIL HAMILTON FAIRLEY, the president, in the chair, a symposium on "daraprim" (pyrimethamine) was held by the Royal Society of Tropical Medicine and Hygiene at Manson House on July 17.

Development of Daraprim

Dr. G. H. HITCHINGS said that the unique biological activity of the 2:4-diaminopyrimidine moiety of daraprim was first recognized in 1947. It and allied substances were found to inhibit the growth of *Lactobacillus casei* in a purine-containing medium. The pyrimidines weighted in

the 5 position were found to be more active than any other of their analogues, and it was predicted that chemotherapeutic agents would be found among them. A structural similarity existed between the diaminopyrimidines and proguanil. Recently it had been shown that proguanil is changed in the body to a much more actively antimalarial substance. This is 1-*p*-chlorphenyl-2:4-diamino-6:6-dimethyl-1:6-dihydro-1:3:5-triazine, and it bears a striking resemblance to daraprim, which is 5-(*p*-chlorphenyl)-2:4-diamino-6-ethylpyrimidine. These compounds, like the dihydrotriazines and phenylpyrimidines, antagonize the action of folic and folinic acids in the growth of the lactic acid bacteria. In the growing rat the toxic effects of daraprim could be alleviated by folinic but not by folic acid, although these toxic effects simulate the effects of folic acid deficiency. The diaminopyrimidines appear to be antimetabolites of the folic acid series of vitamins.

Mr. I. M. ROLLO stated that the action of nearly 300 of the 2:4-diaminopyrimidines had been tested on *Plasmodium gallinaceum* and *P. berghei*, and daraprim had been the most active of these. It effectively prevented the development of exo-erythrocytic forms of *P. gallinaceum* in chicks, though not those of *P. cynomolgi* in monkeys. Small changes in molecular structure drastically affected the antimalarial activity of substances in the series. The most active compounds were all 2:4-diaminopyrimidines substituted in the 5 and 6 positions. Many others with alternative groups in the 2 and 4 positions had been tested and found less active. Resistance to daraprim could be produced in *P. gallinaceum* and *P. berghei* by prolonged deliberate under-treatment with doses having a just appreciable effect in reducing parasitaemia. In mice, however, with heavy *P. berghei* infections, resistance appeared after a relatively small number of treatments. In treating human malaria, therefore, a sufficient dose should be given to eradicate completely and promptly all erythrocytic parasites. In certain cases daraprim had been found almost as active against proguanil-resistant strains in the laboratory as against normal strains.

Clinical Trials

Dr. L. G. GOODWIN said that no toxic effects had resulted from doses of 50 mg. daraprim taken twice weekly for three months by 14 human volunteers. Dr. H. M. Archibald had been the first to try the drug against human malarial infections. A single dose of 5 mg. had been sufficient to cause disappearance of asexual parasites of *P. falciparum* and *P. malariae* from African schoolchildren. The effect was equivalent to that of 300 mg. of mepacrine or "chloroquine," though it developed rather more slowly. Clinical trials in the Gambia, Tunisia, Indo-China, and Assam had shown that single doses varying from 20 to 100 mg. had been effective in treating infections with *P. falciparum*, *P. malariae*, and *P. vivax*, though occasional failures had been encountered. There had been no apparent action on the gametocytes of *P. falciparum*, and that on those of *P. vivax* had been weak. Shute's findings, however, suggested that female gametocytes were affected. In Malaya, Drs. Field, Edeson, and Wilson had been dissatisfied with daraprim because it had acted slowly, some patients having severe symptoms for three days after the dose. It had failed to eradicate asexual *P. falciparum* parasites in 13 out of 80 cases, and increasing the amount of drug given had not greatly improved the proportion of successes. There had, however, been no failures among 22 patients infected with *P. vivax*, and no evidence was found of cross resistance between daraprim and proguanil. As a malarial suppressant a dose of 5 mg. daily was adequate. Following a trial in the Belgian Congo, Vincke had concluded that an annual course of treatment with daraprim early in the rainy season might help to eradicate malaria in wide areas of the district. It was suggested that the principal use of the drug would be as a suppressant in non-immunes and in the mass-prophylaxis of indigenous populations.

Pharmacology

Regarding pharmacology, Dr. Goodwin stated that in experiments with dogs daraprim had caused megaloblastic anaemia, inhibition of leucopoiesis, and degenerative changes in the intestinal epithelium—effects similar to those produced by aminopterin and other antagonists of the folic acid system. In monkeys similar bone marrow changes occurred, together with interference in maturation of the lymphatic tissue, adrenals, and basal layers of the transitional epithelium of the renal pelvis and bladder. Larger doses of drug were needed to produce these changes than those used in human malaria—that is, 10 mg. per kg. per day, which is equivalent to 500–700 mg. per day to a human adult. It was stated that Jacobson had shown that folinic acid is essential in cell division for the shedding of ribonucleoprotein by chromosomes when they separate in metaphase. Antagonists such as aminopterin prevent this process and daughter nuclei fail to form. In view of the observations of McGregor and Smith, it seemed probable that a similar process occurred to the malarial schizont in the presence of daraprim.

Discussion

In the discussion following these papers Dr. C. L. OAKLEY said that 10 mg. per kg. daraprim given daily to monkeys caused changes resembling those produced by anti-folic-acid substances. He showed slides demonstrating these changes.

Brigadier J. S. K. BOYD referred to a communication from Dr. G. R. Coatney describing experiments on volunteers from the Federal Penitentiary, Atlanta, U.S.A. These were bitten by *P.-vivax*-infected mosquitoes and were then given varying doses of daraprim. In those receiving 25 mg. weekly overt malaria did not occur, and had not so far developed during the 251 days since administration of the drug was stopped. From this it was concluded that daraprim probably destroys the liver forms of *P. vivax*.

Dr. DEAN A. SMITH said that his observations supported Jacobson's suggestion concerning folinic acid and cell division. He had noted abnormal chromatin division in *P. falciparum* and *P. malariae* parasites in patients being treated with daraprim.

Dr. T. WILSON stated that in view of his experience in Malaya already referred to he considered daraprim was there unlikely to replace chloroquine in the treatment of the acute attack. He would be reluctant to use it as a suppressant till more was known about the development of resistance to the drug by strains of parasites. In mass suppression adequate supervision of individuals was difficult, and therefore conditions suitable for the production of resistant strains might occur.

Dr. W. JACOBSON said that daraprim was probably not incorporated into the citrovorum factor.

Sir GEORGE MCROBERT described experiences at the Hospital for Tropical Diseases, London, and said that parasites of the Korean strain of *P. vivax* had been found in the blood of patients six and a half days after commencing treatment with daraprim.

Mr. P. S. SHUTE said that in mosquitoes fed on individuals taking daraprim female gametocytes failed to develop into mature oocysts. To study only exflagellation of the male failed to reveal the most important effect of the drug on the mosquito phase of the parasite.

Sir NEIL HAMILTON FAIRLEY stressed the fact that different strains of malaria parasites responded differently to antimalarials. There was, he said, much similarity between the action of proguanil and daraprim in that both arrested the development of malaria parasites at the immature schizont stage. Subinoculation experiments, using proguanil, had shown that such forms were non-infective though they might circulate for many days. He said that as small doses of daraprim prevented overt vivax malaria which would then develop when administration of the drug ceased, daraprim probably acted on the exoerythrocytic and not the pre-erythrocytic forms of *P. vivax*.

Correspondence

Because of the present high cost of producing the Journal, and the great pressure on our space, correspondents are asked to keep their letters short.

Trials of Antihistaminics

SIR,—Like Dr. H. Herxheimer (July 26, p. 228) I have a little experience with antihistaminics and, like him, too, thought it unfortunate that so much ingenuity and time should have been spent on a project (Sevitt *et al.*, July 12, p. 57) that did not and could not decide the question at issue. But I would like to go further than Dr. Herxheimer and make some remarks of a general kind about trials of antihistaminics—remarks that seem the more called for by reason of the very wide range of potencies possessed by the different drugs in this group, and the seeming unwillingness of people to take account of this.

It is, of course, desirable that any therapeutic trial should be well designed and executed and the data properly assessed and interpreted. While much more care is usually given to these matters now than formerly, there are two defects that may sometimes be evident in otherwise irreproachable trials. The first is a failure to state precisely the question that the trial is set to answer. The second is that the answer itself—especially if it is a negative one—instead of being set forth as a particular proposition, is often given a general form that is quite unwarranted. (And even if the investigator makes neither of these mistakes he may be misquoted by others and so made to seem guilty of one or both of them.)

Thus in a trial with a given antihistaminic a very relevant part of the question that should be posed at the outset is: "Does drug X (in dose *y*, *n* times a day) affect the course of the condition?" And the most that the answer could show would be whether this seemed to be so or not. To generalize from the outcome of a single trial with one drug to the class of antihistaminics as a whole is of questionable validity when the results are highly favourable, but utterly indefensible if the drug seems to be of no value at all.

In a trial to test whether antihistamine action *per se* is of benefit in any particular condition a primary essential is to make sure that the drug chosen, its dose, and the spacing of this are such as will in fact produce an antihistamine effect (and, one might add, that the effect is maintained and is of adequate degree). There is little doubt that in some otherwise excellent trials this elementary condition has not been fulfilled. When the results of such trials are negative they are valueless, and if expressed in a general form they are misleading as well. Indeed, the only negative evidence that would seem to justify a general negative conclusion would be that from several trials in each of which it was unquestionable that the drugs had been used in effective doses.

Nor is there much excuse to-day for investigators failing to use effective drugs in effective doses. Pharmacologists have gone to some trouble to determine, in man, the relative potencies and durations of action of different antihistaminics. This information is readily accessible, and it seems a pity that it should so often be ignored—especially when this may lead to the unwitting choice of the weakest available drug for an extensive and painstaking trial.

If antihistaminics are to be tested in a condition not known to respond to them, then, even if the available pharmacological information is appreciated and used, it is surely a good plan to find out by a preliminary or pilot trial whether a large-scale rigorous trial is likely to be worth while. If a small pilot trial, using one or more potent drugs in adequate doses, gives no evidence whatever of any benefit from the treatment, it is probably fair to say that an extensive rigorous trial is not worth while. If, on the other hand, the results of the pilot investigation are obviously favourable, then some of the best conditions applicable to the rigorous trial are already known, and others can be determined by further pilot observations, with consequent saving of time and effort in the design, conduct, and assessment of the main trial. It seems remarkable that pilot trials are not more popular, for their utility and value as a means of getting information that can be used in a large trial are often very great.

These various points may, indeed, be obvious ones; but they all seem, at one time or another, to have escaped the notice of some workers on antihistaminics, and so no apology is needed for drawing attention to them.—I am, etc.,

Department of Pharmacology, Leeds. W. A. BAIN.

SIR,—Dr. S. Sevitt and his colleagues (July 12, p. 57) have ably proved the ineffectiveness of "antistin" on burns when taken systemically. However, my colleagues, W. K. Slack and Edda Boesen (August 11, 1951, p. 360), employed a local application of 2% compresses with the idea of producing a maximum concentration of the drug with minimum side-effects. Others have similarly used a cream base (D. A. Macpherson, July 28, 1951, p. 236). Such a method may prove more fruitful of study by the Birmingham Burns Unit.

At the time it was observed that these drugs possessed a mild antibacterial activity, and an attempt was made to investigate these more fully. Unfortunately circumstances permitted of only a limited series of experiments. It is perhaps opportune to record here a few of the results for the benefit of those interested.

By the plate diffusion technique, using 8-mm. punched holes in a nutrient agar plate, sealing the base with agar, and filling the holes with 2% aqueous solution of various antihistamines, the following zones of inhibition in millimetres were obtained:

Test Organism	"Benadryl"	"Anthisan"	"Thephorin"	"Antistin"	"Pyribenzamine"	"Phenergan"
Oxford staph. + four other strains	7	0	8	5	2	8
β/haem. str., *Str. viridans*, *Str. faecalis*	7	4	8	7	5	9
Pneumococci	7	2	8	6	5	8
C. diphtheriae	7	7	10	6	7	10
Bact. coli	4	1·5	4	4	3	3
Proteus	4	1	6	4	3	7
Pyocyaneus (five strains)	—	—	Slight	—	—	Slight
B. friedländeri (K_1)	4	3	7	7	4	7
Salm. typhi	7	3	7	7	4	6
Sh. sonnei	4	2·5	7	6	3	6

Using the tube technique with doubling dilutions in 1-ml. quantities of 0.5% glucose broth, and seeding with one drop of a 10 times diluted overnight broth culture of the Oxford staphylococcus, the following observations were made after 24 hours' incubation.

Antihistamine	Dilution (in g./100 ml.)									
	2	1	½	¼	⅛	1/16	1/32	1/64	1/128	1/256
Benadryl	−	−	−	−	−	−	+	+	+	+
Anthisan	−	−	+	+	+	+	+	+	+	+
Thephorin	−	−	−	−	−	−	−	+	+	+
Antistin	−	−	−	−	−	−	+	+	+	+
Pyribenzamine	−	−	−	−	+	+	+	+	+	+
Phenergan	−	−	−	−	−	−	−	−	+	+

+ = growth. − = no growth.

Similar experiments with *B. friedländeri* (K_1 strain) gave identical results with benadryl and antistin, but displayed a greater inhibition by two tubes with anthisan and one tube with each of the other drugs.

The presence of 15% serum in the tubes made no difference to the results, but with 5% serum agar plates the zones were 1–2 mm. smaller. Solutions kept for six months at room temperature showed only slight deterioration. The degree of inhibition varies from almost none in the case of anthisan to moderate in the case of phenergan and thephorin and apparently effective against both Gram-positive and -negative organisms alike, the glaring and disappointing exception being *Ps. pyocyanea*. Other investigations, done mostly with benadryl, such as the Rideal Walker phenol coefficient (of 7) and viability counts, brought forth no very interesting features.

This weak effect is, however, quite sufficient for these drugs to act as antibacterial agents on topical application. J. M. Brewster and L. A. Dick (*Tex. Rep. Biol. Med.*, 1949, 7, 69) have even claimed a bacteriostatic effect in blood by oral administration. Little work has been done on this aspect and then mainly with fungi (L. E. Carson, C. C. Campbell, *Science*, 1950, **111**, 689). It may well repay further study.

I would like to record my thanks to the various chemical firms for their liberal supplies of drugs, and to C. A. Abbott for his valuable assistance in these and many unrecorded experiments.

—I am, etc.,

R.A.M.C., Tidworth. O. A. N. HUSAIN.

Future of Public Health

SIR,—In your issue of July 26 (p. 215) Professor A. Leslie Banks is reported as saying that the hospital is now the focal point of the Health Service. Is this to be accepted in so far as care of child life and health is concerned? Can there be any doubt that the home and only the home must be the focal point of any service concerned with the care of infant and child health? Paediatric hospital care exists to assist and not replace or compete with the services provided by the family doctor and the local health service; and to combine with the family doctor and the environmental services in advancing knowledge of the child in health and disease.

No matter the trend in the National Health Service as a whole, it is essential that services for the care of the child should be protected from dominance by a purely hospital outlook. It is asking too much of human nature to entrust boards and management committees responsible for the provision and administration of hospitals intended in the main for adults with the promotion of a service for children which is based on the home and in which hospital provisions form only a subsidiary, no matter how essential and valuable, part.

Professor Banks recommends merging the child welfare services with the hospital and paediatric services. Your leading article on the future of the public health service (p. 203) refers to the place of the general practitioner and to the educational work undertaken by the health visitor. I would submit that the real need in the interest of the child is not merging of the environmental child health services with the hospital services, but even closer association than now generally exists of the environmental services with the work of the family doctor. Difficulties arising in the past from a too arbitrary differentiation between curative and preventive considerations have been largely overtome. It is to be hoped that these will not be revived by the renewed emphasis on "purely educational" measures. Education for health no less than prevention does in fact enter into the daily practice of many family doctors. One of the stumbling-blocks in the way of the greater collaboration urged is the doubts which exist as to what in the future are to be the exact duties of the health visitor. These uncertainties were evident at the Health Visitors Section of the recent Annual Health Congress of the Royal Sanitary Institute. Early resolution of these doubts is needed if children are to derive the maximum benefit from the work of doctors and nurses in the field, and if the home is to retain its rightful place as the focal point in the care of child life and health.

—I am, etc.,

Leeds. W. S. CRAIG.

Training for District Nurses

SIR,—It is very disturbing to learn that the Associations of the County Councils and the Municipal Corporations, and the L.C.C., have stated that they do not consider even a minimal standard of training is necessary for district nurses, or any qualifying examination. These bodies have been responsible for this important branch of the N.H.S. only since the appointed day, and have taken over a specially trained service run by the Queen's Institute of District Nursing, with its magnificent tradition and 62 years of experience. It is to be regretted that the first indication of this responsibility appears to be a retrograde step, and it would be interesting to know if general practitioners, who, after all, work with these nurses, have been consulted.

Surely it is understood that a nurse, working without the constant supervision and help available in hospital, needs some extra experience and qualification, as do others working in the domiciliary field. If the advice of these august but inexperienced bodies is taken, a first-rate service which is the one help the overburdened practitioner can look to will deteriorate, and, to the detriment of the patient, Sarah Gamp may return to the scene once more.—I am, etc.,

Plymouth. O. LL. LANDER.

W.H.O. and Birth Control

SIR,—Dr. H. B. Hewitt's letter (July 12, p. 95) requires an answer because it confuses the issues. Birth is not in the same group of natural events as "epidemics, famine, and human diseases." The suggestion that Catholics could look upon prevention of the last three in the same way as they look upon artificial prevention of childbirth can only be arrived at by a gross error of logic. Perhaps less charitably it might be suggested that an Aunt Sally has been raised simply for the pleasure of knocking it down.

May it be clearly stated, Sir, that Catholics believe that artificial interference with the natural event of birth is wrong for specific reasons connected with the nature of man and the function of sex? They do not believe that the prevention of "epidemics, famine, and human diseases" is wrong just because these are natural events. The negroes are a curly-haired race, but not all curly-haired men are negroes. It is surprising to see such a piece of false reasoning in a scientific journal.—I am, etc.,

London, W.1. F. HARWOOD STEVENSON.

SIR,—Dr. E. L. Sturdee (July 12, p. 95) implies that there is no moral distinction between the practice of contraception and the slaughter of the aged. Surely the distinction is that, while contraception may or may not be a sin, murder is a crime. For this purpose I define a crime as an act which infringes the rights of others: a sin as an act which, while not infringing the rights of others, is nevertheless wrong. If it is agreed that a certain act is not a crime, then the question of whether or not it is a sin is purely a matter of opinion.

If A holds that a certain act is sinful, and B holds that it is not, then, provided that act is not a crime, B has a right to practise it and to enable others to do so; and A, while retaining his right to conduct his own life as his conscience dictates, has no right whatever to obstruct B.

The practice of contraception cannot be regarded as a crime, unless one holds it to be an infringement of the rights of the potential individual represented by the separated sperm and ovum. But in this case it would be as much a crime to abstain from sexual intercourse whenever the opportunity offered as to use a contraceptive appliance.—I am, etc.,

London, S.W.7. D. A. T. TIZARD.

SIR,—Dr. Agnes Sadler (July 12, p. 95) is curiously defeatist when she writes that war, disease, and famine are inevitable features of our life on this planet. At present they are certainly the three major means by which Nature limits world population. The new methods for the eradication of many killing diseases are pre-eminently responsible for the rapid increase of world population, an increase that must *ultimately* result in the failure of world food resources to yield adequate supplies. If, as most of us hope, further wars are to be avoided, then of necessity it must be famine that will wield the shears that cut short the threads of our lives. Is it not pertinent to consider the degrees of unpleasantness involved in death from war, disease, and famine? Surely the long-protracted misery of empty bellies, the cruel degradation of a starving people, far outweigh in horror death from disease or from war. If this is so,

must we not admit that the advances of medicine may prove to be a snare and a delusion? We will have preserved many millions from their natural fate, only to find a far worse one awaits their children. Is it not the responsibility of the medical profession to try to avert such a disaster?—I am, etc.,

Guy's Medical School, S.E.1. ANDREW MALLESON.

⁂ This correspondence is now closed.—ED., *B.M.J.*

Malaria in Britain

SIR,—Further to the letters by Professor G. Macdonald (July 12, p. 92) and Dr. P. H. Round (July 26, p. 227) it may be interesting to record a case of malaria treated recently. The patient was a 32-year-old ex-soldier who, until his demobilization from Korea in the middle of November, 1951, had been taking prophylactic mepacrine. He arrived in England on December 18, but had no symptoms whatever until the second week of June, 1952, when he began to feel feverish and complained of lassitude on alternate days. His symptoms apparently were thought trivial enough for him to be put down as a neurotic, but on admission to hospital on July 22 a routine blood film showed the gametocyte stage of *Plasmodium vivax*. At no time during his war service in Italy and Egypt or in Korea did he have any symptoms suggestive of a malaria attack.—I am, etc.,

Harold Wood, Essex. J. E. JELINEK.

⁂ Malaria in Britain is discussed on page 347.—ED., *B.M.J.*

Malaria Control in Mauritius

SIR,—Since the preparation of my article on the Malaria Eradication Scheme in Mauritius, which is published in today's issue (p. 309), there has been a new development which has considerably altered future policy. Following the success of larviciding in one district, this method was extended to the whole of the coastal zone during the off-season of 1951 in an attempt at species eradication of *A. gambiae*. At first the results appeared to be satisfactory, but, after unusually heavy rainfall in February of this year, *A. gambiae* breeding increased, particularly on the west coast of the island, in spite of all attempts to control it.

At this stage, therefore, two alternative courses were open. First, by increasing the field personnel and modifying the method, it would be possible to intensify the attack in an all-out attempt at elimination of *A. gambiae*. This would entail a considerable increase in annual expenditure which would have to be maintained for three or four years. It was considered that such a project would have a fair chance of ultimate success, but no guarantee could of course be given.

A study of the malaria situation during 1952 revealed some interesting facts. The notifications by dispensaries and hospitals suggested that the disease had fallen to a negligible level, as is demonstrated in the following table:

1948	32,913
1949	18,944
1950	4,121
1951	1,000
1952	27

Note.—1. Blood slides were obtained from all the 27 cases notified so far in 1952, and on examination gave without exception a negative result.

2. During the three months May–July, 1952, nil returns were submitted from all dispensaries and hospitals as against 11,936 cases in the same period of 1948.

During the first seven months of 1952, in a specific search for malaria cases, the unit laboratory has examined some 20,000 blood slides. Only 34 positive cases have been found, many of which from the history were probably relapses of the disease.

It would thus seem that transmission of malaria has virtually ceased throughout the island, in spite of the presence of large numbers of vector mosquitoes. In the absence of *A. funestus*, originally the principal vector of the disease in Mauritius responsible for maintaining all-the-year-round transmission, the problem is a purely seasonal one. No specimen of *A. funestus* has been found, in spite of continual intensive searching, for more than two years.

In the absence of malaria, it was not considered to be economically sound to embark on a large-scale campaign aimed at the eradication of a species which was not maintaining transmission. The second alternative, therefore, was to introduce a control scheme which would continue indefinitely on a diminishing scale. As long as the unit is staffed permanently by a small scientific and laboratory staff there will be no danger of a breakdown of the control. In the absence of malaria carriers among the human population there can be no transmission of the disease. All positive cases found are therefore being treated and followed up after one, three, and six months. It is considered that by selective house-spraying with residual insecticides the annual cost of the scheme can be reduced at least to the equivalent of 9d. per head of population. A close watch must of course be maintained on the reintroduction of human carriers and also of fresh anopheles species which may complicate the picture.

As long as a scientific staff remains in the control unit a new focus of fresh transmission, should it develop, can be discovered and obliterated long before it can attain even mild epidemic proportions. It has been demonstrated conclusively elsewhere that residual insecticides and other auxiliary measures can adequately control malaria even under hyperendemic conditions. The control scheme in Mauritius starts with one conspicuous advantage: there is virtually no malaria.

Thank you for according me this opportunity of bringing my article up to date.—I am, etc.,

Floreal, Mauritius. M. A. C. DOWLING.

Pain in Childbirth

SIR,—I would like to thank Dr. D. J. Adams Clark for his very restrained letter (July 26, p. 229). May I at the same time assure him that I used no such term as "callous indifference," nor did I refer in any way at all to general practitioners? I am afraid that the Press report, which published the above phrase in inverted commas, thereby implying a quotation, was entirely inaccurate in its usual sensational manner.—I am, etc.,

London, W.1. F. NEON REYNOLDS.

Gastric Gossip

SIR,—Sir Arthur Porritt (July 19, p. 107) rightly draws attention to the difficulty in assessing the results of surgery in peptic ulcer, and while so many surgeons persist in regarding peptic ulcer patients as lesser breeds of men, and their complaints after gastrectomy as tiresome manifestations of neurosis, this state of affairs is likely to continue. Sir Arthur, for instance, appears to hold the view that fear of an ulcer accounts for that series of mechanical, nutritional, and metabolic disturbances nowadays included under the term "the dumping syndrome." No doubt he would have us believe that the men and women whose small intestines are not constructed to cope with the influx of large amounts of undigested food, unmixed with bile or pancreatic secretion, are really suffering from a "lack of integration."

If psychogenic factors are as important as Sir Arthur suggests, surely he should make a plea for less surgery, and not for more. And if he believes that there are only 5% of failures, why does he lament the passing of the operation of gastro-jejunostomy?

Writing in the *Lancet* recently (July 5, p. 44), Mr. Michael J. Smyth observes that it is remarkable how many doctors there are, afflicted with duodenal ulcer, who are content to have medical treatment for their relapses rather than face the prospect of a gastrectomy. Perhaps they have seen the "skeletons in the cupboard."—I am, etc.,

Harrogate. A. J. GLAZEBROOK.

Peptic Ulcer in the East

SIR,—I should like to comment on two points from Sir Arthur Porritt's lucid survey of the peptic ulcer position ("A Gastric Gossip," July 19, p. 107). He states, when discussing aetiological factors: "It (peptic ulcer) is a rare condition in the black races and in Asiatics." I have not long returned from Borneo, after spending three years in an area with a good sample of Asian races, predominantly Chinese, Malays, and "Dayaks" (using the word to cover the indigenous people other than Malays). Although I would endorse his statement with regard to the Malays, my impression was that the Chinese are at least as ulcer-prone as people in England. Overall statistics might mislead, as

these stoical people are not wont to seek medical aid without impelling symptoms. I have, however, seen more cases of perforated peptic ulcer than strangulated hernia in Chinese, while haematemesis or melaena due to peptic ulcer was comparatively common and seen in both sexes (males predominating).

The condition was not so commonly seen in the Dayak races, but the majority of these people lived some travelling time away in the jungle, and only severe symptoms—perhaps coupled with advice from a roving missionary—would cause them to make the journey to hospital. I twice had to intervene for organic pyloric stenosis, considered to be due to peptic ulceration, in primitive Dayaks—one a Kelabit woman and the other an Iban man. One of these had made a four-week journey to reach the hospital.

Sir Arthur's further statement, that a man transferring his habitat from temperate to tropical regions thereby considerably reduces the chance of his developing an ulcer, is interesting. Although the European population in our area was insufficiently large, and also, too, selected medically, for statistical comparison, it was not uncommon to see peptic ulceration arisen *de novo* in this group, and the occasional relapse in a former ulcer patient from a temperate climate. —I am, etc.,

London, S.E.1. MICHAEL FLINDT.

Sympathectomy for Raynaud's Disease

SIR,—In their interesting article on this subject (June 28, p. 1377) Messrs. J. B. Kinmonth and G. J. Hadfield claim that the results of ganglionectomy and preganglionic operations have never before been adequately compared. But in my Hunterian Lecture (Haxton, 1947) I presented a comparison substantially similar to theirs, though on a smaller number of cases, and reached the same conclusion that there is no real difference between the late results of the two types of operation. Some degree of relapse is unfortunately common to both, but nearly all patients maintain that they have been benefited, some considerably, by the operations.

There is a regrettable tendency in their article and other recent papers (Haimovici, 1951; Jepson, 1952) to ignore the undeniably strong evidence in favour of regeneration after sympathectomy. The facts are:

1. Regeneration occurs readily in cats and dogs, even over wide gaps in the sympathetic chain.

2. Sympathetic activity reappears in the upper extremity after months or years of complete absence following sympathectomy.

3. In both ganglionectomy and preganglionic section cases this returned activity can be temporarily abolished by procaine-diodone paravertebral block in the upper thorax below the level of the second rib, with no sensory or motor disturbance, and no Horner's syndrome in the preganglionic section cases (Haxton, 1947). I have demonstrated this repeatedly, and it can be confirmed by anyone sufficiently adept at paravertebral needling. In the only case in which it was attempted a prolonged restoration of the hand to its post-operative warmth and dryness was achieved by injection of aqueous phenol (Haxton, 1949) in the same locality.

I have encountered no explanation other than regeneration which will explain these findings, and would be interested if any could be proffered. Certainly they rule out the possibility of the returned activity being due to pathways and intermediate ganglia in the cervical region. If regeneration is to be accepted I do not see the necessity to postulate that in ganglionectomy cases preganglionic fibres must grow down into the upper limbs. Postganglionic fibres are also divided when the chain is sectioned, and there is no reason to suppose that these cannot find their way along the upper limb nerves. Certainly the latent period of absent sympathetic activity is much longer (sometimes years) after ganglionectomy than after preganglionic section, and this is much in favour of regeneration, a longer period being required for growth of the fibres down the extremity. The point made by Kinmonth and Hadfield, that if regeneration occurs it should do so more readily after Telford's than after Smithwick's operation, is not supported by my tests with procaine-diodone paravertebral blocks. These indicate that in man, as in dogs, cats, and monkeys, the main sympathetic supply to the upper limbs comes from below the third thoracic segment and is thus little affected by avulsion of the second and third thoracic nerve roots.

It is true that some severe Raynaud cases show relapse within a few days of complete operation, and these must have a marked local sensitivity of the digital vessels to cold, as postulated by Lewis. However, in cases of later relapse the reappearance of symptoms often coincides with the return of vasoconstrictor activity (Simmons and Sheehan, 1939). The implication that sympathetic activity may play an important part in attacks of Raynaud's phenomenon is supported by the finding that such attacks in the hands may appear for the first time after, or may be greatly aggravated by, thoraco-lumbar sympathectomy for hypertension, even though the blood pressure does not remain lowered. The attacks must here be attributed to compensatory increase in vasoconstrictor activity in the upper part of the body where the sympathetic pathways remain intact.

I must say, finally, that I agree that the preganglionic type of operation is the better for the upper limb, since it avoids unpleasant side-effects, and that Telford's is the best operation for the expert operator to use, as it causes much less pain and discomfort to the patient than the posterior approach. This we have proved by carrying out the two operations on opposite sides in the same patient and inviting comparison.—I am, etc.,

Manchester. H. A. HAXTON.

REFERENCES

Haimovici, H. (1951). *Angiology*, **2**, 423.
Haxton, H. A. (1947). *Brit. J. Surg.*, **35**, 69.
—— (1949). *British Medical Journal*, **1**, 1026.
Jepson, R. P. (1952). *Ann. roy. Coll. Surg. Engl.*, **9**, 35.
Simmons, H. T., and Sheehan, D. (1939). *Brit. J. Surg.*, **27**, 234.

Treatment of Intersex

SIR,—Your insistence on brevity in published articles (not so apparent in the correspondence columns) has exposed me to justifiable criticism on account of my failure to discuss, in my article on intersex (June 14, p. 1264), the effects of castration in the group I designated intersex males with purely feminine external genitalia. Mr. Michael C. Oldfield (July 26, p. 228) does me less than justice, however, in accusing me of overlooking the case originally published by Chapple (*British Medical Journal*, 1937, **1**, 803), since an explicit endeavour was made to limit the bibliography to papers appearing after that date, and the circumstances of this case had so recently received a lengthy recapitulation from Mr. Oldfield himself at the Plastic Surgeons Conference.

The object of my review was to lift the discussion of intersex problems above the anecdotal level to which it is so often relegated, and in assessing the prognosis of the various types I had therefore to supplement my own experience by reports from the literature. Thus, so far from "condoning wholesale castration" in this group of intersex males, I stated that "treatment is scarcely necessary in many cases," adding in view of the numerous records that "most have required bilateral orchidectomy because of pain."

With regard to the carcinogenetic potentialities of stilboestrol, the danger appears to have been somewhat overestimated. Although many hundreds of thousands of elderly men have received prolonged dosage with this drug, there are, I believe, no more than 15 examples of complicating mammary cancer on record. Moreover, Campbell and Cummins (*Cancer*, 1951, **4**, 303) have produced good evidence that some at least of these were in fact metastatic tumours, and not related to the oestrogen therapy.—I am etc.,

London W.1. D. INNES WILLIAMS.

POINTS FROM LETTERS

Wound Healing in Anaemia

Mr. G. H. COLT (London, W.1.) writes: The delay in the healing of aseptic wounds in patients suffering from the anaemia of malaria (see also *Journal*, July 26, p. 228) caused the authorities to stop the performance of many operations of election in such cases as hernia and haemorrhoids occurring in the Salonika command during the first world war. The tissues failed to unite even after three or four weeks. This was a common experience throughout the command.

Obituary

GEORGE CARMICHAEL LOW, M.A., M.D., C.M. F.R.C.P.

The death of George Carmichael Low on July 31 at the age of 79 marks the end of the formative era of tropical medicine in Britain. As the disciple of Sir Patrick Manson he was in at the beginning, and he was present at the solution of many of the fundamental discoveries of what then constituted a new science; it was upon him that the mantle of Manson fell to continue the traditions of the London School of Tropical Medicine. His passing therefore removes a figure and a character that was well known and everywhere recognized as original.

George Carmichael Low was born in 1872 at Monifieth, Forfarshire. He thus inherited some Scottish traditions and was brought up in a strictly disciplined school, so that this training became apparent in his mannerisms throughout his long life. After schooling at St. Andrews he studied medicine in Edinburgh, qualifying with first-class honours in 1897 and proceeding M.D. with gold medal in 1910. He also obtained the gold medal in tropical medicine in 1912. Among the great teachers of his day he placed Greenfield very high. The fame of Manson was then in the ascendant, and Low came to London to sit at his feet. Soon he travelled to Heidelberg and Vienna to learn fresh techniques, especially the celloidin method of cutting sections of insects which Manson wished to apply to a collection of filaria-infected mosquitoes sent to him by Thomas Bancroft, of Brisbane, the son of the great Joseph, after whom *Filaria bancrofti* had been named by Cobbold. Returning to London in 1899, he was not long in showing almost at the first attempt, and for the first time, that the larval filaria emerged from the tip of the proboscis and that man became reinfected by the mosquito in the *act of biting*. This solution ran contrary to Manson's hypothesis, which envisaged the worm re-entering the human body via drinking-water in which the mosquito vector had died. This discovery, published in the *British Medical Journal*, 1900, **1**, 1456, naturally attracted much attention, and it was not long before further outlets for his energy were forthcoming.

In the autumn of 1900 Low was engaged in fundamental malaria research. Consequent upon Ross's original discoveries regarding the mosquito transmission of malaria and Grassi's confirmation (1899) in man, Manson found that in official circles scepticism about this important question still existed. A scheme was therefore devised to demonstrate that Europeans could live and remain free of fever in the most malarious districts, provided they slept at night in a mosquito-proof house. Low was placed in charge and had as his companions L. W. Sambon and Signor Terzi, the well-known artist. These three lived three months from August, 1900, in a screened hut erected at Ostia in the Roman Campagna. During the daytime they roamed about the countryside amidst the fever-stricken peasants, collecting mosquito larvae and making observations, but being hustled with typical Scots discipline into their hut every night by Low. The experiment was absolutely successful, entirely vindicating Manson's hypothesis, and pointed the way to badly needed anti-mosquito measures on the West Coast of Africa and elsewhere. During this time Low's little malaria squad was visited by the King of Italy, while Professor Grassi came there to collect *Anopheles maculipennis* larvae, hatched out in Rome and set to feed on malaria cases. They were then dispatched to Manson in London to bite his son, P. T. Manson, who subsequently developed benign tertian malaria (*British Medical Journal*, 1900, **2**, 949). His next expedition was as Craggs scholar to the West Indies in 1901, where he travelled widely, eventually reaching British Guiana. There he studied *Filaria demarquayi* among the aboriginal Carib Indians of South America.

The year 1903 was spent with the Royal Society's Commission in Uganda, where, together with Castellani and Nabarro, he was investigating the aetiology of "sleeping sickness." He gave most attention to the possible role which *Filaria* (now *Acanthocheilonema*) *perstans* might play in this disease, a hypothesis which Manson himself had put forward.

A new superintendent of the newly created London School of Tropical Medicine was required, and Low was the obvious choice. Thereafter he found himself chained to the metropolis, spending the rest of his career in the service of the school and as physician to the Hospital for Tropical Diseases. He followed the school from the Albert Docks to its present situation in Bloomsbury. Throughout this long period, some 34 years before he retired, he taught assiduously and examined both for the London M.D. and the D.T.M.&H. of the Conjoint Board. His clinical afternoons were always well attended by large and critical classes. They became known as fountains of instruction and as a source of no little amusement. At various times Low served on committees of the Colonial Office and of London University. Possibly one of his greatest achievements, and certainly, as a loyal pupil of Manson, one of which he was most proud, was the institution of the Royal Society of Tropical Medicine and Hygiene. He was an original founder (with Sir J. Cantlie) in 1907, and served as secretary from 1912 to 1920—a most critical period—and as president from 1929 to 1933. History will recall that it was mainly through his efforts that the society was housed in its present quarters in Manson House, Portland Place, in 1930. As the result of his original works he was honoured by being appointed Membre d'Honneur of the Société Belge de Médecine Tropicale and a corresponding member of the French Société de Pathologie Exotique. In 1929 he was awarded the Mary Kingsley Medal of the Liverpool School of Tropical Medicine.

Low's contributions to medical literature were many; and in the textbooks his partnership with Sir Neil Fairley in *Price's Textbook of Medicine*, and his article with Clifford Dobell on amoebiasis in Byam and Archibald's *Practice of Medicine in the Tropics*, are best known.

No account of Low's life would be complete without a reference to his contributions to scientific ornithology. He had a particularly tidy mind which, when bent to the collection of scientific data, shrank from no labours. He was scrupulously accurate and painstaking. A good bird-watcher, he did much to enhance our knowledge of the birds of London, and became in later years as well known on the London reservoirs and on the banks of the Thames as in the Bird Room at South Kensington. He was a valued member of the council of the Zoological Society and of the Royal Society for the Protection of Birds. For many years he was secretary and treasurer of the British Ornithological Club and editor of the *Bulletin*. He was remarkably successful

in keeping the younger ornithologists together. He made some very valuable contributions to serious ornithological literature in his monograph *The Literature of the Charadriiformes*, a second edition of which appeared in 1931, and in his stupendous account of *Aves* in the centenary volume of the Zoological Society in 1929.

Most people found in Low a kindly, entertaining soul with an attraction especially for young people, dogs, and children. There were those almost unconscious family Scottish witticisms. The erect and possessive presence, the kindly greeting of "How's yoursel'?" the recurrent nod of the head, the clipped staccato speech, the wise twinkle of one eye, the smile which accompanied authoritative and stentorian aphorisms—these will long be remembered, as also a kind of "spooneresque" unconscious wit which fell from him during the course of his public appearances. Low married in 1906 Edith Nash. There were no children of the marriage, and much sympathy will go out to her, his lifelong companion.

LESLIE CUNNINGHAM, M.A., M.B., B.Ch. F.R.C.P.

In the untimely death on July 30 of Dr. Leslie Cunningham, Liverpool lost a distinguished physician. Educated at Cambridge and St. Bartholomew's Hospital, Leslie Cunningham was a Foundation Scholar at Downing College. He qualified with the conjoint diploma in 1916 and took the M.B., B.Ch. in 1921. In 1923 he obtained the M.R.C.P. and in 1942 was elected F.R.C.P.

After serving in the R.A.M.C. with the rank of captain during the first world war, Cunningham went to the Northern Hospital, Liverpool, and in spite of his considerable war experience as a medical officer he started at the bottom again in the post of house-physician. With his outstanding ability and charm of personality he passed rapidly through the stage of registrar to become honorary assistant physician and later honorary physician. Among his other appointments he was honorary consulting physician to Leasowe Children's Hospital, Ormskirk General Hospital, and Birkenhead General Hospital.

E. N. C. writes: Cunningham's first and chief love was the Northern Hospital. His loyalty was outstanding, and there he made many friends and no enemies. He constantly attracted students, who spoke with delight of his careful teaching, his gentle sense of humour, and his kindness. These qualities were well known to his friends and colleagues, but they were evident no less to the young.

He was one of those physicians who, though familiar with the rapid scientific advances of our art, was at his best as a clinician, drawing on a great knowledge of medicine and with wisdom of a rare kind. To many of us he would seem to embody the virtues which Hippocrates preached, and many students must have gained something of this spirit from their contact with him. His widespread practice from North Wales to the Lake District is a testimonial to the esteem in which his character and knowledge were held by general practitioners, and patients had implicit faith in his skill.

Perhaps the key to the man is to be found in his family, where he found such happiness with his devoted wife and children. His recreation at the end of a long day was to amuse the children, and no one could be more entertaining in his droll and kindly way. Sometimes his self-effacement might suggest to the uninformed a certain lack of interest or acumen, but when he gave judgment on any problem, whether clinical or administrative, it was clear that he had a deep understanding of the matter before he spoke. His opinion was correspondingly sure of attentive hearing in both his hospital and his practice. His complete selflessness led to that consideration for other people's troubles which is such an important attribute in a good doctor. By his character and example he has lit a flame in Liverpool which will burn for many years and has handed a torch to the young practitioners of medicine which they will carry on to others of succeeding generations.

Sir HUGH CAIRNS, K.B.E., D.M., F.R.C.S.

The death of Sir Hugh Cairns was recorded in the *Journal* of July 26 (p. 233).

J. M. W. writes: I should like to pay a tribute to the work done by Sir Hugh Cairns for the Army. Before the second world war Cairns was consultant in neurosurgery to the Queen Alexandra Hospital, Millbank. On the outbreak of war one of his first enthusiasms was the mobile neurosurgical unit. After much intensive work and trial he devised a motor vehicle to carry everything necessary for emergency head surgery. The vehicle carried operating-table, instruments, dressings, and the staff of neurosurgeons, physician, anaesthetist, general duty officer, and sisters. The first unit arrived in France in May, 1940, but, most unfortunately, was captured complete (with the exception of the sisters) in the very early operations. This was a sad blow to Cairns. However, more units were mobilized and more specialist personnel assembled. These did very good work in North Africa, Sicily, Italy, and North-west Europe.

Cairns was most interested in motor-cycle accidents, and worked out an excellent type of crash helmet which was universally adopted in the Service and undoubtedly saved many lives. He was also a pioneer in the use of penicillin in head wounds, and I well remember his visit to North Africa with Sir Howard Florey, and his enthusiasm and work there. He was also able to see the work of the mobile neurosurgical unit at first-hand. Sir Geoffrey Jefferson has well said that Cairns usually got what he wanted. If Hugh Cairns thought that a thing was essential he would not take no for an answer, and by his pertinacity and schemes some way would be found of surmounting every difficulty.

I whole-heartedly agree with all that has been written by abler pens than mine about Hugh Cairns. As a colleague and a man he was a stimulating experience to work with, and his energy and enthusiasm were unbounded. His death leaves a deep sense of personal loss. The Army owes him a very great debt.

CATHERINE CHISHOLM, C.B.E., M.D., F.R.C.P.

Dr. Catherine Chisholm's death was recorded in the *Journal* of August 2 (p. 287).

M. M. B. and E. P. write: Swiftly the sorrowful news that Catherine Chisholm had died went abroad and we mourned the loss of a well-tried friend, a beloved and revered colleague, a distinguished citizen of Manchester, and a pioneer medical woman of this country. Flags were flown at half mast at the University of Manchester and at the Duchess of York Hospital for Babies.

Catherine Chisholm's father was a medical graduate of the University of Edinburgh; her mother a Lancashire lady of mill-owner stock. Blended and at times warring elements of her being came from this union of Scotland and England. The woman we knew and loved was the product, big in stature and in mind, lovable, warm-hearted, level-headed, yet impetuous; a great friend, a colleague to respect and to be reckoned with, and in time of need a tower of strength. To her father she was indebted for that far-seeing and liberal outlook which encouraged her first to take an Arts degree at Manchester University and later to apply successfully for permission to study medicine. In both courses she distinguished herself, gaining the Bishop Lee Greek Testament Prize in her Arts course and several prizes in her final year in medicine. After graduating in medicine in 1904 she spent some time in resident posts, then settled for a number of years in general practice in Manchester. Perhaps "settled" is a wrong description of such a dynamic personality; the years in practice, however, served to show her the real voca-

tion she sought, which was the care of children. To see a desired objective was to Catherine Chisholm synonymous with achieving it. The high mortality rate in Manchester from summer diarrhoea in infants led to the founding in 1914 of a 12-cot hospital—predecessor of the Duchess of York Hospital for Babies—in a converted house in Chorlton to combat the disease. In this new venture Catherine Chisholm was supported by many Manchester men and women, medical and lay, notably by James Niven, the medical officer of health, and Lorrain Smith, the professor of pathology. At the Hospital for Babies Catherine Chisholm acted as mother and mentor, and the hospital, her staff, her colleagues, and her committee responded with loyalty and love. Nothing but the best was good enough for the babies. From her travels and her wide reading she sifted new ideas which could be adopted. Early in the history of B.C.G. inoculation she endeavoured to start an experimental study of its efficacy. To her disappointment the project was turned down. She lived to see B.C.G. adopted as an efficacious prophylactic. For years before the war she was an advocate of breast milk for sick and premature infants, and had instituted residential nursing mothers at the hospital. Early in the war this idea was expanded to a scheme for the collection and sterilization of breast milk from the homes of nursing mothers. From this has stemmed the flourishing milk bank, which supplies not only the Hospital for Babies but is available for sick babies elsewhere. Even the details of child care received her attention. She loved children, and with her insight she realized that none of the scientific advances she advocated in the care of infants were complete unless they were accompanied with affection. In her preoccupation with the Hospital for Babies and the children's departments of Hope Hospital and the Northern Hospital she did not lose sight of the larger issue of the training of students, medical and nursing, in the care of children. She continually pressed for the creation of a chair in child health in the University of Manchester, not only to stress the importance of the subject but to associate all the aspects of the medical care of children in the region round Manchester. The appointment of a professor in child health in the University in 1947 was a great joy to her. Catherine Chisholm had been ill for a year before her death. To an impatient, vital person, this increasing limitation of her activity was a supreme trial. The open countryside she loved was too far away, and books, her greatest solace, were too heavy, but she still had her friends to bring her news of the world. She retained her amazing sense of humour and to the last she was cared for lovingly by her devoted companion. One of Catherine Chisholm's greatest characteristics was her immense generosity. What she gave to her patients, to her colleagues, and to her adopted city are her lasting memorial.

C. L. C. writes: This is no appraisement of the great work that Catherine Chisholm has done for her patients, for the advancement of women, and for her personal friends, but only a note on her origins and early days. Catherine Chisholm entered the Owens College Medical School, Manchester, 54 years ago, after taking her B.A. degree. She was greatly devoted to her Highland father. Dr. Kenneth Mackenzie Chisholm was the son of a Gairloch forester who had the ability and determination to take a medical degree at Edinburgh. He took his pretty young Lancashire wife up to Scotland to practise, but finding that she was miserable away from her own people came down to Lancashire again and settled at Radcliffe for his life's work. From his square brick house, surrounded by a garden which occupied his leisure hours, he drove round to his patients in a pony trap and ruled his family and surroundings with a resolute autocracy. He gave his two daughters a good education, and Mrs. Chisholm saw to it that they were skilled in domestic work and regular in religious observance. There were loud but unavailing protests from them one Sunday afternoon because Mrs. Chisholm recognized some Chopin they were playing as a waltz and promptly forbade it. No dance music was to be played on Sunday. Church attendance, daily prayers, and grace before meat were all indispensable; the slightest deflection from the beaten path was at once stopped and punished. Yet I do not think anyone who knew Catherine in later years would think that this strict rule had diminished her independence of will or thought, and it certainly did not prevent an affectionate devotion to her parents.

Dr. GEORGE MITCHELL, who died at his home at Insch, Aberdeenshire, on July 19 at the age of 68, was born at Old Rayne, Aberdeenshire, on March 10, 1884, the son of Dr. Patrick Mitchell, and was educated at Gordon's College, Aberdeen, and at the University of Aberdeen, where he graduated M.B., Ch.B. in 1907. After holding the appointment of house-surgeon at Aberdeen Royal Infirmary he became an extern clinical assistant at the Coombe Lying-in Hospital, Dublin, and while there he took the L.M. in 1908. He settled in practice at Insch in 1909, and remained there until his death. During the war of 1914–18 he was in charge of V.A.D. hospitals in Scotland. For many years a member of the British Medical Association, Dr. Mitchell was chairman of the Aberdeen and Kincardine Counties Division of the Association from 1936 to 1938, and was president of the Aberdeen Branch for the same period. He also represented his constituency at the Annual Representative Meeting at Newcastle-upon-Tyne in 1921.

We are indebted to Dr. A. U. Webster for the following appreciation: In sorrow we note the death of Dr. George Mitchell, for, with his passing, not only has a great and well-known general practitioner gone from us but his death has also meant the end of an era in medical practice. Dr. Mitchell came of a medical family; his father and his grandfather both practised in that same delectable part of Aberdeenshire, the Gairloch. He became house-surgeon to the late Mr. Scott Riddell, and even then he showed himself by his work to be far above his fellows. Interested in surgery, which was so well taught in his day, he did original work with blood counts in acute abdominal conditions when such work was still in its very early stages, and had he cared he could easily have obtained high rank as a consultant and general surgeon. But the call of general practice and the country was too strong, and after a course of study at the midwifery schools of Dublin he settled in practice among his own people, remaining with them for over 40 years. His reputation as a general practitioner was unrivalled and his success was never challenged. His love for surgery never left him, and, owing in great measure to his drive and foresight, a war memorial cottage hospital was established in Insch after the war of 1914–18. It it, along with the work of a heavy general practice, he did a great amount of general surgery, and he was recognized by his fellow practitioners as an operator of the first rank. Keen on many aspects of country life, he was an excellent horseman and a well-known and sound judge of saddle horses. He is survived by Mrs. Mitchell and two daughters and one son, now an orthopaedic surgeon, who had an outstanding record in the last war.

Dr. STEPHEN YOUNG BAIRD, who died in London on July 18, was educated at the University of Glasgow, graduating M.B., Ch.B. in 1935. He obtained an appointment as medical officer to a tea estate in Bengal, in India, in 1937, and while there he acted as honorary secretary of the Northern Bengal and Dooars Division of the British Medical Association in 1938–9. During the last war he served in the R.A.M.C. from 1940 to November, 1945, when he was released with the rank of captain. In 1946 he became medical officer to a tea estate in Assam.

We are indebted to Dr. W. J. SAVAGE, president of the Assam Branch of the B.M.A., for the following appreciation: It came as a great shock to medical officers in the tea industry in Assam when it became known that Dr. Stephen Young Baird had died in a London nursing-home. He was an energetic worker, and had an extremely sound knowledge of tropical medicine. He did some useful work in the prevention and treatment of kala-azar. He had been

attached to the tea industry since 1937, and had distinguished Army service from 1940 to 1945, when he resumed practice in the Takurbari district on the north bank of the Bramaputra. He was well loved by his patients and friends and respected by his colleagues. By nature he was entirely unselfish and straightforward, and had a ready wit. A keen and sound member of the B.M.A., he was never known to miss an annual general meeting of the Assam Branch. For some years he led his district on the polo field, and was recognized as one of the better players in Assam. The untimely death of this skilful and popular practitioner is a great loss to the profession and a grievous blow to his many friends and patients. He is survived by his young widow and two children, to whom we extend our deepest sympathy.

Medical Notes in Parliament

Judgment on Durham

Sir WALTER MONCKTON announced on July 30 the award of the Board of Arbitration appointed to determine the difference between the Durham County Council and the Joint Emergency Committee of the Professions. He said the House would recall that differences between the county council and some of its professional employees had persisted for a long time. The present difference had arisen over the county council's regulation that applications for extended sick pay must be made by or through a trade union or other appropriate organization. The assistance of his Department had been sought in June after some 4,000 teachers employed by the county council had handed in notice of resignation from August 31. At the request of the parties he had appointed a Board of Arbitration to determine the difference. The parties undertook in advance to accept and implement any award made by the Board. Both parties accepted that, so far as certain professions were concerned, membership of a trade union or professional organization should not be imposed as a condition of employment. Accordingly the agreed terms of reference included the question of whether the present regulations of the council governing the making of applications for extended sick pay were in conflict with the principle of voluntary membership of a trade union or professional organization and should be withdrawn.

The Board's award, Sir Walter Monckton said, was that in so far as the regulations required such applications to be made through a trade union or other appropriate organization they were in conflict with the principle and should be withdrawn.

Mr. ALFRED ROBENS asked whether the House could accept the situation as being finally closed. Sir WALTER said he hoped and thought so.

Drinks in Hospital

Mr. JAMES HUDSON on July 31 asked the Minister of Health to insist that provision of alcoholic beverages to patients in hospitals should not be allowed except under doctor's prescriptions.

Mr. IAIN MACLEOD said it was the practice to advise hospital authorities that alcoholic beverages should be supplied to hospital patients as part of their diet only on doctors' orders. Their cost was not recorded separately, but he had no reason to think that they were being supplied extravagantly.

Powers of Hospital Consultative Committee

On July 31 Mr. C. F. H. GOUGH inquired how far hospital joint staffs consultative committees were competent to discuss and decide upon matters of hours and wages.

Mr. IAIN MACLEOD replied that rates of pay and conditions of service generally in the hospital service were determined nationally by the Whitley Councils and were outside the competence of hospital staffs consultative committees. They might, however, consider purely local matters, such as the distribution of the working hours of particular grades of staff in their own hospitals.

Medical Examination of Z Men

Colonel J. H. HARRISON asked whether the Minister of Labour had arranged for ex-Japanese prisoners-of-war, called up for further service, to have special consideration at their medical examination.

Mr. HAROLD WATKINSON replied on August 1 that Sir Walter Monckton had sent a note to chairmen of medical boards early in March last, asking examiners to pay particular attention to the medical history of these reservists, and to take full account of any condition or disability resulting from imprisonment still present and of any possibility of the reservist's physical or mental condition deteriorating as a result of privations suffered as a prisoner-of-war.

Bone-conduction Hearing-aids.—The Electro-Acoustics Committee of the Medical Research Council has assessed the results of clinical trials of an experimental bone-conduction aid produced for the National Health Service, and has recommended this aid for issue to patients for whom a bone-conduction aid is more suitable than an air-conduction aid. Mr. Macleod will arrange large-scale production as soon as possible.

Child Psychotherapists.—There is no agreement at present as to what should be the qualifications and functions of non-medical personnel engaged in psychotherapy, or under what conditions they should be employed in the Health Service, if at all.

Hospital Waiting-lists.—Patients in England and Wales awaiting admission to hospitals taken over under the National Health Service Act numbered 503,584 on December 31, 1951.

On August 1 both Houses adjourned until Tuesday, October 14.

Medico-Legal

HYPNOTIST'S APPEAL

[FROM OUR MEDICO-LEGAL CORRESPONDENT]

The Court of Appeal on July 30[1] allowed part of the appeal of Ralph Slater, the hypnotist, from the judgment of Mr. Justice Croom-Johnson at Sussex Assizes on March 30, 1952.[2] The Court set aside the order for £1,000 general damage and £107 special damages awarded to the plaintiff, Miss Diana Grace Rains-Bath, on the grounds of Mr. Slater's negligence, and ordered a new trial of that issue. It did not disturb Mr. Justice Croom-Johnson's judgment for £25 damages on the issue of assault.

In his judgment Lord Justice Singleton said that the plaintiff's case had been that she had been told under hypnosis that she would be very frightened and would cry for her mother. The medical evidence was that it was foolish and wrong for a hypnotist to put into the mind of someone under hypnosis anything in the nature of terrifying thoughts, and that if suggestions of such a nature were put into the mind of a subject great care must be taken to remove any trace of fear. The defendant's case was that he had been in no way negligent, that as a stage hypnotist he had hypnotized possibly 25,000 people without any complaint, and that he had not told the plaintiff that she would be frightened.

It was obvious that there were serious considerations for the jury. The medical witnesses must have found some difficulty in connecting the condition of the plaintiff 18 months after the hypnosis with the hypnosis itself, though there was some evidence on that. It was important that it should be put before the jury with clarity. There was nothing in the summing-up which directed the jury to the question whether the defendant had implanted in the mind of the plaintiff something of a terrifying nature, and, if he

[1] *The Times*, July 31.
[2] *British Medical Journal*, April 12, p. 823.

had done, whether that was something wrong and dangerous, and whether he had taken sufficient steps to remove it.

The judge's directions on these matters were not enough, and therefore the case made was not before the jury as it should have been, and on that issue there must be a new trial.

Lord Justice Birkett and Lord Justice Morris delivered concurring judgments.

Universities and Colleges

ROYAL COLLEGE OF PHYSICIANS OF LONDON

At a quarterly comitia of the College held on July 31, with the President, Sir Russell Brain, in the chair, it was announced that the Duke of Edinburgh had graciously accepted the Honorary Fellowship of the College.

The following Fellows of the College were elected officers for the ensuing year: *Censors*, A. H. Douthwaite, C. F. T. East, F. P. Lee Lander, J. R. H. Towers; *Treasurer*, W. G. Barnard; *Registrar*, Sir Harold Boldero; *Harveian Librarian*, E. C. Dodds, F.R.S.; *Assistant Registrar*, M. I. A. Hunter.

Dr. George Graham was appointed Harveian Orator and Dr. E. J. Wayne Bradshaw Lecturer, both for 1953. The following lecturers were also appointed: Lumleian (1953), Dr. E. A. Carmichael; Goulstonian (1953), Dr. D. A. K. Black; FitzPatrick (1953), Dr. M. Davidson; Oliver-Sharpey (1953), Professor C. Wilson; Lloyd-Roberts (1953), B. Ifor Evans, D.Lit.; Humphry Davy Rolleston (1953), Professor R. A. McCance, F.R.S.; Croonian (1954), Dr. J. Forest Smith; FitzPatrick (1954), Dr. C. E. Newman.

Sir Russell Brain was reappointed as the College representative on the governing body of the British Postgraduate Medical Federation, and Sir William Hume on the council of King's College, Newcastle-upon-Tyne, in the University of Durham.

The Murchison Scholarship was awarded to Dr. C. W. Crane.

Membership

The following candidates, having satisfied the Censor's Board, were elected Members of the College: J. H. Biggart, M.D., U. Birnbaum, M.B., P. S. Brown, B.M., H. M. T. Coles, M.D., W. M. Dixon, M.B., E. Dresner, M.D., D. Emslie-Smith, M.B., D. M. D. Evans, M.B., K. T. Evans, M.B., S. G. Faris, M.B., J. M. Farrar, M.B., D. D. Felix-Davies, M.B., I. T. Gabe, M.B., C. D. Gettliffe, M.B., R. R. M. Harman, M.B., Lieutenant, R.A.M.C., R. A. K. Harper, M.B., R. P. Jayewardene, M.B., P. K. Krishnankutti, M.D., J. P. R. MacFarlane, M.B., T. McKeown, M.D., Cynthia J. McPherson, M.B., D. Mendel, M.B., T. G. Milliken, M.D., D. Mustafa, Diploma, Kitchener School of Medicine, Khartoum, D. G. Neill, M.B., K. Rajasuriya, L.M.S., A. E. A. Read, M.B., A. C. Ronald, M.B., J. D. Sheehan, M.B., R. G. Shorter, M.B., J. P. Stanfield, M.B., P. G. I. Stovin, M.B., C. G. A. Thomas, B.M., G. W. Thorpe, M. B., A. L. Turnbull, B. M., W. Van't Hoff, M.B., R. H. C. Wells, M.B., D. M. Williams, M.B., P. O. Williams, M.B., M. F. T. Yealland, M.B.

Licences

Licences to practise were conferred upon 129 candidates (including 25 women) who have passed the final examination in Medicine, Surgery, and Midwifery of the Conjoint Board and who have complied with the necessary by-laws: V. Ainsworth, A. L. Bass, I. S. Batey, K. B. Bender, A. Betts, M. H. H. Bishop, June E. Brett, J. R. Brown, J. W. Brown, C. C. Bull, G. M. B. Bulman, A. R. Butterfield, G. R. C. Campion, H. Caplan, D. A. Chiswell, J. A. Clappen, B. J. F. Coles, H. A. Collins, Betty R. Corbin, H. J. E. Cox, H. F. Crawford, G. T. Crook, R. L. E. Cumberbatch, Bethan Davies, E. P. Denton, J. M. Dolphin, Aileen B. Donnison, A. K. Dowle, M. Dulake, W. F. Dummer, A. V. Dunlop, B. A. East, T. A. Evans, N. H. Eve, F. W. Fairhead, K. T. Farn, P. M. Forster, J. L. Fraser, A. G. E. Fung-a-Fat, D. R. Gamble, Mary I. Gibbons, B. Gilbert, I. L. Glaisher, H. W. Goldfarb, J. H. Goode, K. Grainger, Bernadette Gregory, Joan E. Hancock, R. B. Handscombe, N. B. Harris, J. D. Harte, W. D. Hayley, D. A. Heath, E. R. Herst, F. A. Hill, C. B. Hodes, Vera M. C. Hollyhock, Blanche M. Hooper, Shirley E. Hoyes, K. R. Hughes, J. P. Hurrell, G. H. Jeavons, R. D. Jones, A. F. Jordan, L. Kader, E. E. F. Keal, D. J. Kidd, D. W. G. King, Iris M. Krass, C. G. Lambert, R. J. W. Lambert, L. H. Lee, F. Lees, K. M. McC. Lewis, H. Lindsay, Patricia M. Lumb, Nancy S. Maguire, G. Mangan, R. D. Mann, J. Manuel, D. H. Marjot, Philippa M. Martin, J. A. Mead, G. W. Middleton, F. Morley, Y. A. Motala, J. H. Muende, G. D. Mulholland, Kathleen M. Neville, Elizabeth C. Nightingale, M. J. O'Neill, W. N. B. Parker, W. P. Pereira, Mary E. R. Perry, R. J. V. Peter, R. A. N. Petrie, E. M. Pilkington, L. J. Posener, L. B. Prescott, M. N. Prichard, Freda E. Prince-White, F. C. Ramsey, G. Ll. S. Rankin, Shelagh M. Richards, G. G. Robbie, T. E. Roberts, Winifred M. S. Robinson, Vera Rose, G. Screech, D. B. Shaw, K. Shiffman, D. N. Sinclair, J. J. Slome, Isabel G. Smith, J. L. Stevenson, Mary L. Stewart, J. L. Szentpétery, Elizabeth A. Thomson, G. N. W. Tilsley, D. Tingle, G. R. P. A. Vernon, P. Wallis, A. W. Warltier, P. A. Watt, Joyce D. Waye, E. A. Wilson, P. G. Woolf, J. G. Wotherspoon, S. S. Young.

Diplomas

Diplomas were granted, jointly with the Royal College of Surgeons of England, to the following successful candidates:

DIPLOMA IN PUBLIC HEALTH.—Margaret A. M. Howard, F. X. Letellier, R. L. Lindon, J. F. McGovern, M. L. Rawal, G. M. Reynolds, Mary M. E. Rutter, M. Shrinagesh, I. H. Syed.

DIPLOMA IN OPHTHALMOLOGY.—N. S. Batheja, R. D. Calcott, B. M. Chatterjee, G. V. Cole, P. H. Cowen, R. Cowley, C. W. L. Crawford, E. B. Doany, Stella P. Eadie, Margaret Ford, H. Fraser, S. D. Gun-Munro, M. Habibulla, J. Hughes, I. G. Johnston, P. M. Joseph, P. J. Mathews, Jean C. I. Melville, Eugenia M. O'Keeffe, J. N. Ormrod, J. A. E. Primrose, A. K. Sinha, D. N. Sud, J. E. Thomas, R. Traill, C. G. Tulloh, Nora P. Woods.

DIPLOMA IN INDUSTRIAL HEALTH.—A. W. Gilks, J. I. Lesh, R. W. McConnachie, S. L. Malhotra, M. L. Rawal, J. F. Skone, W. C. Turner, M. D. Warren.

Diplomas in Anaesthetics (*Journal*, July 5, p. 47, and July 26, p. 239), in Pathology, in Medical Radio-diagnosis, and in Medical Radiotherapy (*Journal*, July 5, p. 47), in Laryngology and Otology, in Tropical Medicine and Hygiene, and in Psychological Medicine (*Journal*, July 26, p. 239), were granted, jointly with the Royal College of Surgeons of England, to the successful candidates whose names were printed in the reports of the meetings of the Royal College of Surgeons of England in the issues of the *Journal* indicated above.

ROYAL COLLEGE OF OBSTETRICIANS AND GYNAECOLOGISTS

At a meeting of the Council of the College held on July 26, with the President, Dame Hilda Lloyd, in the chair, the following officers were elected: *President*, Mr. Arthur A. Gemmell; *Vice-presidents*, Professor Gilbert I. Strachan and Mr. A. J. McNair; *Honorary Treasurer*, Mr. A. C. H. Bell; *Honorary Secretary*, Mr. Humphrey G. E. Arthure; *Honorary Librarian*, Mr. A. J. McNair; *Honorary Curator of the Museum*, Mr. Aleck W. Bourne; *Honorary Treasurer, Australian Regional Council*, Professor Bruce T. Mayes; *Honorary Treasurer, New Zealand Regional Council*, Mr. H. K. Pacey.

Sir Stanford Cade was invited to attend meetings of Council as representative of the Council of the Royal College of Surgeons of England.

The following were elected to the Membership: J. D. Arora, Lily Arratoon, J. L. M. Bean, A. C. Bhoumik, C. H. Borsman, G. H. Burgess, G. G. Champion, S. Chinnatamby, G. R. Clare, J. F. Correy, N. K. Crooke, G. E. Cummins, A. M. Dawson, R. D. De Vere, D. T. Dodds, C. P. Douglas, R. Dowie, S. K. Dutt, Aileen C. Foley, M. S. Forrest, D. F. P. Gordon, W. J. Gordon, J. J. Handler, A. Hunter, Daphne M. E. Kayton, I. A. McDonald, Margaret W. Macgregor, T. P. Magee, T. McG. McNie, A. G. Mathew, P. R. Myerscough, J. G. O'Sullivan, Marjorie M. Paterson, A. Persey, Evelyn Peters, G. W. Prueter, D. Puflett, Marie Quadros, M. B. Quane, B. B. Rakshit, H. Rees, D. Seif-Eldin, C. A. Simmons, E. L. Trott, Mary E. Walker, R. A. Warren, H. B. Watson, J. A. Williams.

CONJOINT BOARD IN SCOTLAND

The following candidates, having passed the final examinations, have been granted the diploma of L.R.C.P.Ed., L.R.C.S.Ed., L.R.F.P.S.Glasg.: R. S. Ahluwalia, Mavis F. Anderson, F. J. Badenhorst, J. B. B. Baird, C. E. Barth, G. A. C. Binnie, E. H. Brown, D. G. Chambers, B. Cornes, W. A. Dalgleish, E. A. Ehlinger, S. Fogel, Brenda Gale, Colina Graham, Ruth E. Graham-Yooll, J. M. H. R. Hardy, A. R. Isaac, A. H. Kassam, V. Kritzinger, C. H. Lalgee, Pamela M. Lambden, P. A. Lowe, W. A. Mahon, Pearl K. Malasky, Christine W. Mann, R. W. S. Miller, E. C. Moffat, J. I. Morrison, J. A. McIlvride, Agnes F. Mackay, A. Orwin, Joyce E. A. Ovens, Hedwig M. M. Papigay, Audrey Pilbeam, Jean M. Price, R. A. W. Ratcliff, Margaret H. Rodger, Enid Sagar, R. Shabetai, Thelma D. Standing, R. Steele, A. Stevenson, C. Ward, M. H. Weinberg, M. M. Wiener.

INFECTIOUS DISEASES AND VITAL STATISTICS

Summary for British Isles for week ending **July 19** (No. 29) and corresponding week 1951.

Figures of *cases* are for the countries shown and London administrative county. Figures of *deaths* and *births* are for the 160 great towns in England and Wales (London included), London administrative county, the 17 principal towns in Scotland, the 10 principal towns in Northern Ireland, and the 14 principal towns in Eire.

A blank space denotes disease not notifiable or no return available.

The table is based on information supplied by the Registrars-General of England and Wales, Scotland, N. Ireland, and Eire, the Ministry of Health and Local Government of N. Ireland, and the Department of Health of Eire.

CASES in Countries and London	1952					1951				
	Eng. & Wales	Lond.	Scot.	N. Ire.	Eire	Eng. & Wales	Lond.	Scot.	N. Ire.	Eire
Diphtheria	25	3	4	0		30	3	16	1	1
Dysentery	140	16	40	0	2	301	81	90	2	
Encephalitis, acute	3	1	0	0		12	0		0	
Enteric fever:										
Typhoid	2	0	1	0	1	5	0	0	2	3
Paratyphoid	39	0	1 (B)	0		37	4	3 (B)	0	
Food-poisoning	180	33		0		188	15		2	
Infective enteritis or diarrhoea under 2 years				5	27				13	37
Measles*	7,225	730	100	164	284	6,306	155	174	177	155
Meningococcal infection	25	1	13	2	1	37	2	17	2	
Ophthalmia neonatorum	34	1	10	0		47	2	13	0	
Pneumonia†	259	16	94	1	1			97		
Poliomyelitis, acute:										
Paralytic	104	4	11	3	2	61	5	15	2	1
Non-paralytic	59	2				73	2			
Puerperal fever§	213	37	13	0				10		
Scarlet fever	1,038	84	138	10	56	830	80	113	16	27
Tuberculosis:										
Respiratory			112	23				133	38	
Non-respiratory			36	7				41	7	
Whooping-cough	2,206	88	145	60	124	3,380	185	294	74	79

DEATHS in Great Towns	1952					1951				
	Eng. & Wales	Lond.	Scot.	N. Ire.	Eire	Eng. & Wales	Lond.	Scot.	N. Ire.	Eire
Diphtheria	0	0		0		*0*	0	*0*	0	0
Dysentery	0	0					0		0	
Encephalitis, acute		0					0			0
Enteric fever	0	0		0		*0*	0	*0*	0	
Infective enteritis or diarrhoea under 2 years	5		1	2		*10*		*3*	1	0
Influenza	4	0		0		*3*	0	*0*	0	0
Measles		0		1			0	*0*	0	0
Meningococcal infection		1	1				0	*0*		
Pneumonia	114	11		14		*104*	13		15	4
Poliomyelitis, acute	4	0				*4*	0			0
Scarlet fever		0		0			0	*0*	0	0
Tuberculosis:										
Respiratory	73	9	13	5		*135*	19	*20*	7	10
Non-respiratory		2	3	0			6	*11*	2	2
Whooping-cough	1	0		0		*3*	0	*0*	0	0
Deaths 0–1 year	199	23	19	7		*198*	23	*29*	10	16
Deaths (excluding stillbirths)	4,023	567	447	104		*3,968*	630	*538*	119	153
LIVE BIRTHS	7,449	1114	814	210		*7,121*	1219	*970*	242	401
STILLBIRTHS	179	32	23			*160*	27	*26*		

* Measles not notifiable in Scotland, whence returns are approximate.
† Includes primary and influenzal pneumonia.
§ Includes puerperal pyrexia.

Vital Statistics

Poliomyelitis

Poliomyelitis notifications in the week ending July 26 (30th week of the year) were as follows: paralytic, 157 (104); non-paralytic, 96 (59); total, 253 (163). This is an increase of 90 compared with the previous week, the figures for which are in parentheses. Up to and including this week the overall notification rate for England and Wales was 3.26 per 100,000.

The total number of cases of poliomyelitis reported in the U.S.A. from April 5 up to July 24 was 5,506, according to the U.S. Public Health Service (*New York Times*, July 25). This figure compares with 5,313 for the same period in 1949, when the last serious epidemic occurred.

Week Ending July 26

The notifications of infectious diseases in England and Wales during the week included: scarlet fever 1,173, whooping-cough 2,040, diphtheria 21, measles 7,363, acute pneumonia 247, acute poliomyelitis 253, dysentery 135, paratyphoid fever 75, and typhoid fever 5.

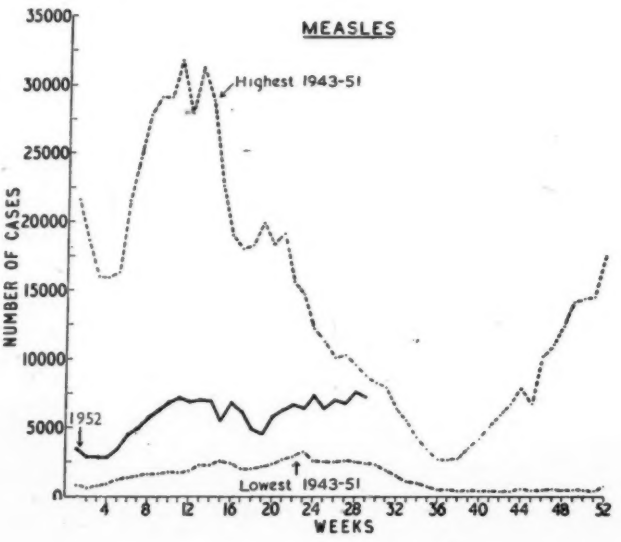
Infectious Diseases

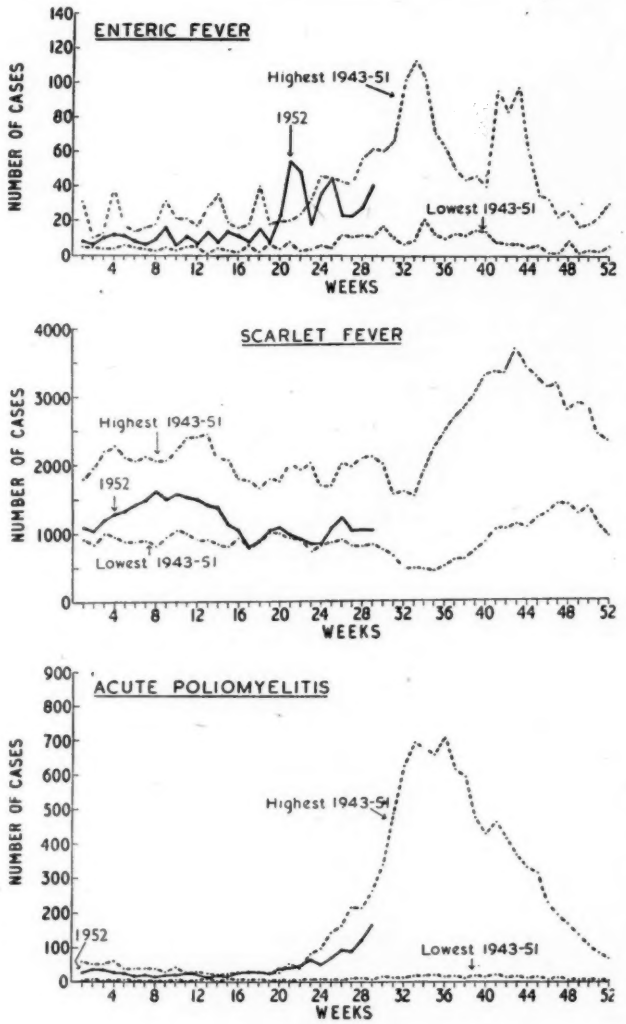
In England and Wales during the week ending July 19 notifications of acute poliomyelitis increased by 44 to 163 and of paratyphoid fever by 14 to 39. Decreases were recorded for measles 316, whooping-cough 36, and dysentery 11.

From Yorkshire North Riding 15 new cases of paratyphoid fever were reported, 12 of them at Whitby U.D. In the Glamorganshire outbreak 6 new cases were recorded, and from Pembrokeshire also 6, 4 of these from Fishguard and Goodwick and 2 from Haverfordwest R.D.

Graphs of Infectious Diseases

The graphs below show the uncorrected numbers of cases of certain diseases notified weekly in England and Wales. Highest and lowest figures reported during the nine years 1943–51 are shown thus - - - - - -, the figures for 1952 thus ————. Except for the curves showing notifications in 1952, the graphs were prepared at the Department of Medical Statistics and Epidemiology, London School of Hygiene and Tropical Medicine.





Population Movement in Scotland

The 1951 census shows that there has been a considerable movement from the countryside to the towns in Scotland in the last 50 years. In 1901, 74.3% of the population were in urban areas; in 1951 the figure was 82.9%. The total population of Scotland also increased during these years by 14% from 4,472,103 to 5,095,969, and in 1951 the urban population was 27.2% greater than in 1901. The central industrial region has been mainly responsible for the increase. The population in rural areas in 1901 was 25.7% of the total population, while in 1951 it had fallen to 17.1%, in spite of the increase in the Scottish population as a whole.

In 1901 there were 153 villages with 1,000 or more inhabitants, compared with 225 in 1951, while in 28 villages the population fell below a thousand between 1901 and 1951. In 1901 burghs with 1,000 or more inhabitants contained 65.1% of the Scottish population, while in 1951 they contained 69.7%.—*Census 1951, Scotland*, Vol. 2 (H.M.S.O.).

Industrial Accidents and Diseases

The number of workpeople (other than seamen) in the United Kingdom whose deaths from accidents in the course of their employment were reported in June was 125, compared with 123 (revised figure) in the previous month and 104 (revised figure) for June, 1951.

Cases of industrial disease reported during the month were as follows: lead poisoning 4, other poisoning 5, anthrax 3, epitheliomatous ulceration 11, chrome ulceration 9; total 32. There were no deaths.—*Ministry of Labour Gazette*, July, 1952.

Medical News

Malaria in Britain.—The letter on malaria in Britain by Professor G. Macdonald (*British Medical Journal*, July 12, p. 92) has resulted in a number of letters asking for further information. Among them was a suggestion that the present Army standard malaria treatment should be published briefly, with a reference to the Refresher Course article by Sir Gordon Covell (October 27, 1951, p. 1021). Army medical authorities stress the importance of early diagnosis and the importance of taking blood films and having them examined without delay. They recommend that quinine and "pamaquin," or chloroquine ("nivaquine" is the British equivalent) and pamaquin, should be used in the case of benign tertian malaria. The following course is the most likely to cure the condition and lessen the chance of relapse:

Quinine bihydrochloride 10 grains (0.65 g.) t.d.s. for 14 days in conjunction with pamaquin 0.01 g. t.d.s. 14 days. If the patient has had a previous attack the length of the quinine-pamaquin course may be extended to 21 days.

As an alternative chloroquine (nivaquine) may be given:

1st day, 0.9 g. chloroquine base, 6 tablets.
2nd day, 0.6 g. base, 4 tablets.
3rd to 5th day, 0.3 g. base, 2 tablets.
Total 16 tablets. Pamaquin should also be given in the same dosage as before.

During this treatment the patient should not be allowed to go about, and is best treated in bed. Malignant tertian malaria without evidence of pernicious manifestations can be treated with the course of chloroquine given above. Pamaquin is not required, as there is little tendency to relapse. However, it is best to have such cases admitted to hospital, as severe or pernicious symptoms necessitate the immediate administration of intravenous quinine. Mepacrine is still used in the routine treatment of uncomplicated M.T. malaria.

1st day, 0.3 g. (3 tabs.) t.d.s.
2nd and 3rd days, 0.2 g. (2 tabs.), t.d.s.
4th to 7th day, 0.1 g. (1 tab.), t.d.s.

"Paludrine" (proguanil) is not used in the treatment of malaria in the Army, although it is extensively used for suppression and has almost completely replaced mepacrine in this respect.

Malaria Control in British Guiana.—The annual general meeting of the Royal Society of Tropical Medicine and Hygiene was held at Manson House on June 19, and after reports of the council and the treasurer had been accepted an address on malaria control in British Guiana was given by Dr. George Giglioli, with a fine series of coloured slides. Dr. Giglioli has been in charge of malaria control work there for over 20 years, in the early part of which malaria was severe and almost universal. The low-lying coastal plain of British Guiana is waterlogged and has a rainfall of something like 100 inches a year; it is covered with sugar cane and rice fields and is intersected by countless irrigation canals arranged symmetrically. In these waters *Anopheles darlingi*, a vigorous carrier of malaria, bred prolifically. The situation has been completely changed solely by the use of D.D.T. as a residual insecticide in dwellings and other buildings. The extent of the change may be inferred from the statement that the last *A. darlingi* was found in 1946, in spite of continued search, which still goes on, and that last year only 24 cases of verified malaria were reported, and these were probably relapses. Yet other anophelines still abound, but they do not seek man or enter his dwellings. They are therefore not affected by the D.D.T. house-spray programme, but this is not important, since they do not attack man and therefore do not carry malaria. *Aëdes aegypti*, the potential vector of yellow fever, has also disappeared. The coastal plain is therefore clear of malaria, but *A. darlingi* exists in the sparsely inhabited interior, and could invade the coast along certain rivers, the waters of

which are favourable to it. Spraying teams now keep the mosquitoes at bay on these rivers, and it is hoped that, by concentrating here and ceasing residual spray on the coast, malaria will be controlled with only 25% of the present effort. It is true that British Guiana is rather a special case, and that *A. darlingi* is almost ideal for control in this way, but these facts do not detract from the boldness of the conception of eradication. Control of malaria has enormously reduced general and infant mortality and morbidity, and no doubt will produce a community more physically fit than ever before, to develop the great and still only partly tapped resources of a great country.

Society for Clinical Pathology in France.—Aiming to link biology and medicine more closely, a society under the presidency of Professor Henri Benard has been founded in France. One of the vice-presidents is Professor Justin Besançon, editor of *La Semaine des Hôpitaux*. The society intends to bring together French doctors interested in clinical pathology, to further work on the subject, and to establish contact with other societies of the same kind abroad. Two or three symposia are planned yearly, the first being scheduled for November 24 this year; the subject will be the porphyrinurias.

Medicine and Physical Education.—The William Hyde Award (£300) for 1950 has been awarded to Dr. R. W. Parnell, of the Institute of Social Medicine, Oxford, in recognition of his work on the application of special physical measurements (somato-typing) to students in Oxford. The award is made from funds provided by the Nuffield Trust in memory of William Hyde, who died in 1945, and whose concern for the future of the hospital services of the country to a large extent led to the establishment of the Nuffield Provincial Hospitals Trust.

COMING EVENTS

South African Medical Congress.—The 1952 Congress will be held in the Central (Arts) Block of the Witwatersrand University, Johannesburg, September 22 to 27. In addition to the numerous scientific sections there will be two plenary sessions, on September 23 (Dr. M. M. Suzman: Clinical Application of Corticotropin and Cortisone Therapy), and on September 25 (Dr. Alice Cox: Psychological Illness in South Africa: the Problems of Treatment and Care). The Medical Association of South Africa extends a cordial welcome to members of the British Medical Association to attend the Congress, and further information will gladly be supplied by the honorary organizing secretary, Dr. J. Gluckman, Congress Office, Medical House, 5, Esselen Street, Hospital Hill, Johannesburg.

Gynaecological Cytology at Brussels.—Professor R. Bourg announces that he is organizing a course of lectures on gynaecological cytology at the Brugmann Hospital, Brussels, in September. The course will run from September 22 to October 2, and will be divided into two parts, elementary and advanced cytology. Registration fee will be 500 Belgian francs, and full details may be obtained from Professor Bourg, Université Libre de Bruxelles, 50, Avenue F. D. Roosevelt, Brussels.

Public Health Courses.—The Royal Institute of Public Health and Hygiene is holding courses for the certificate and diploma in public health and the diploma in industrial health. Full information, entry forms, and prospectuses may be obtained from the secretary of the Institute, 28, Portland Place, London, W.1.

Fourteenth International Congress of Military Medicine and Pharmacy.—For the first time the organization of a congress of this nature will be held in a South American country—at Montevideo, Uruguay, March 1 to 6, 1953. Full information may be obtained from Dirección General del Servicio de Sanidad Militar, 8 de Octubre y Mariano Moreno, Montevideo, Uruguay.

International Gynaecological Meeting.—The National Union of French Gynaecologists and Obstetricians is organizing an international meeting of national obstetrical and gynaecological associations in Paris in May, 1953. The questions to be discussed are the protection of mothers and newborn infants in wartime, and training in gynaecology and obstetrics. Full details from Dr. Jacques Courtois, 1, Rue Racine, Saint-Germain-en-Laye (Seine et Oise).

APPOINTMENTS

EAST ANGLIAN REGIONAL HOSPITAL BOARD.—*Assistant Chest Physician at Peterborough Chest Clinic*, R. D. Chalmers, M.R.C.S., L.R.C.P. *Anaesthetic Registrar at United Norwich Hospitals*, W. J. Love, M.B., B.Ch.

FARQUHAR, J. V. L., M.R.C.S., L.R.C.P., D.P.H., Assistant Senior Medical Officer, Liverpool Regional Hospital Board.

HOSPITAL FOR SICK CHILDREN, Great Ormond Street, London, W.C.—*Part-time Registrar to Department of Psychological Medicine*, J. R. E. Dobson, M.B., Ch.B., D.P.M. *Assistant Resident Medical Officer, Tadworth Court*, Catherine N. McRobert, M.B., B.S. *House-physicians*, H. E. F. Davies, M.D., Joan C. Wells, M.D., M.R.C.P. *House-surgeons*, O. W. Davies, M.B., B.S., R. L. Batten, M.R.C.S.

MCCOUBREY, A. F., M.B., Ch.B., D.P.H., Medical Officer of Health for Counties of Roxburgh and Selkirk.

MANCHESTER REGIONAL HOSPITAL BOARD.—*Consultant General Surgeon, South Cheshire Hospital Centre*, H. Hassall, M.B., Ch.B., F.R.C.S. *Consultant Anaesthetist to Thoracic Surgery Team, Blackpool*, C. P. Brown, M.B., Ch.B., D.A. *Assistant Pathologist, Withington Hospital, Manchester*, T. A. White, M.B., Ch.B., D.C.P. *Assistant Pathologist, Salford*, H. B. Marsden, M.B., Ch.B., D.C.H. *Assistant Radiologist to Macclesfield and South Cheshire Hospitals and Parkside Hospital, Macclesfield*, E. A. Beetles, M.S., M.B., D.M.R.D. *Assistant Anaesthetist, West Manchester Hospitals*, Marjory A. Pollock, M.B., B.Ch., D.A. *Assistant Anaesthetist, Oldham Hospitals*, Ilse R. Hadelmayr-Kuhn, M.D., D.A.

NORTH-EAST METROPOLITAN REGIONAL HOSPITAL BOARD.—*Maximum Part-time Thoracic Surgeon (Consultant), North-east Metropolitan Hospital Region*, M. Bates, F.R.C.S. *Full-time or Maximum Part-time Anaesthetist (Consultant), Oldchurch Hospital*, H. Howell-Jones, M.R.C.S., L.R.C.P., D.A. *Part-time Surgeon (Consultant), Metropolitan Hospital*, G. M. Lunn, M.Chir., F.R.C.S. *Part-time Anaesthetist (Consultant), Whipps Cross Hospital*, J. W. Monro, M.B., B.Chir., D.A. *Maximum Part-time Obstetrician and Gynaecologist (Consultant), Oldchurch Hospital*, E. E. Philipp, F.R.C.S., M.R.C.O.G. *Part-time Ear, Nose, and Throat Surgeon (Consultant), Rush Green Hospital*, C. S. Savage, F.R.C.S. *Part-time Anaesthetist (Consultant), Broomfield Hospital*, S. D. K. Stride, M.R.C.S., L.R.C.P., D.A. *Part-time Paediatrician (Consultant), Harold Wood Hospital*, F. S. W. Brimblecombe, M.D., M.R.C.P., D.C.H. *Part-time Dermatologist (Consultant), Oldchurch Hospital*, E. L. Cohen, M.B., B.Chir. *Full-time Psychiatrist (Consultant), Brentwood Mental Hospital*, J. R. P. Edkins, M.D., D.P.M. *Full-time Anaesthetist (Consultant), Chase Farm and other hospitals within the Group*, P. W. S. Gray, M.B., B.S., D.A. *Part-time Dermatologist (Consultant), Whipps Cross Hospital*, L. Sefton, M.R.C.P. *Part-time Ear, Nose, and Throat Surgeon (Consultant), Mile End and St. George-in-the-East Hospitals*, M. Marks, M.R.C.S., L.R.C.P., D.L.O.

O'GRADY, TERENCE P., M.B., B.Ch., D.P.H., Divisional Medical Officer, Division No. 13, Lancashire County Council.

PLEYDELL, MALCOLM JAMES, M.C., M.D., D.P.H., Deputy County Medical Officer of Health for Northamptonshire.

ROADS, PETER G., M.D., D.P.H., Medical Officer of Health, Borough of Shrewsbury, and Assistant County Medical Officer for Salop.

WHITE, GEORGE MALCOLM JAMES, M.B., Ch.B., House-surgeon, North Ormesby Hospital, Middlesbrough.

WOLFF, F. W., M.B., B.S., Medical Registrar, West Suffolk General Hospital, East Anglian Regional Hospital Board.

BIRTHS, MARRIAGES, AND DEATHS

BIRTHS

Bihari.—On July 15, 1952, at University College Hospital, London, W.C., to Mac, wife of Dr. Julian Bihari, a daughter—Katherine Anne.

Gilks.—On July 29, 1952, at St. Mary's Hospital, Paddington, London, W., to Nancy (formerly Franklin), wife of Dr. A. W. Gilks, M.R.C.S., L.R.C.P., D.P.H., D.T.H., a son—Nicholas.

Lange.—On July 27, 1952, at New End Hospital, Hampstead, London, N.W., to Joyce, wife of Mr. M. J. Lange, F.R.C.S.Ed., a son.

MARRIAGES

Herdman—Howarth.—On July 30, 1952, in London, Gawin J. S. Herdman, M.B., to Betty E. Howarth, M.R.C.P.

DEATHS

Browne.—On July 24, 1952, in Dublin, O'Donel Thornley Dodwell Browne, Litt.D., M.A.O., F.R.C.P.I., F.R.C.O.G.

Church.—On July 30, 1952, at Torquay, Anna Lydia Church, M.D.

Cunningham.—On July 30, 1952 Leslie Cunningham, M.B., F.R.C.P., of the Manor House, Prenton, Cheshire, and 72, Rodney Street, Liverpool.

Ellacombe.—On July 13, 1952, at 53, Alma Road, Rosebank, Capetown, South Africa, Gilbert Henry Wemyss Ellacombe, M.R.C.S., L.R.C.P., aged 83.

Jennings.—On July 24, 1952, in a London hospital, Dorothy Margaret Jennings, M.B., Ch.B., D.R.C.O.G., of 707, West Derby Road, Liverpool, aged 27.

Low.—On July 31, 1952, at 7, Kent House, Kensington, London, W., George Carmichael Low, M.D., F.R.C.P., aged 79.

Any Questions?

Correspondents should give their names and addresses (not for publication) and include all relevant details in their questions, which should be typed. We publish here a selection of those questions and answers which seem to be of general interest.

Admission of Poliomyelitis Cases to General Wards

Q.—*Should cases of infantile paralysis be admitted to general wards during the acute stage of the disease?*

A.—The modern treatment of the acute stage of poliomyelitis requires that many resources which are often available only in a general hospital should be at the disposal of those who are treating the patient. For example, a bronchoscopy or tracheotomy may be necessary, or a team of highly skilled nurses may be required to deal with bulbar paralysis or the complicated drill of handling a patient in a respirator. These facilities are, unfortunately, only rarely available in the under-staffed fever hospitals in this country, and many physicians feel that poliomyelitis cases should sometimes be admitted to general hospitals. During an epidemic a special ward may be set aside for such cases in general hospitals, or side rooms may be used, and when this can be done the risk of infection, which is in any case slight, becomes quite insignificant. In general it may be said that, in deciding where the patient should be nursed, the interests of the patient should take priority over the theoretical risk involved in sending the patient to a general rather than an isolation hospital.

Sun-glasses

Q.—*Is there any harm in allowing children to wear sun-glasses, or should they be encouraged to accustom their eyes to normal bright light when young?*

A.—The eyes were evolved to adapt themselves to normal bright sunlight, and there is no reason why children—or, for that matter, adults—should wear sun-glasses unless in conditions where there is an unusual amount of reflected sunlight (as at the seaside), or in circumstances where a bright sun is being faced for long periods (as in driving a car).

Aortic Incompetence in Marathon Runner

Q.—*A fit, active male of 22 years with an excellent cardiac reserve (he competes successfully in 20-mile marathons) is found at routine examination to have a marked aortic diastolic murmur, conducted down the sternum. He had an attack of chorea at 9 years; his heart is not enlarged; his blood pressure is normal; and his W.R. is negative. What is the prognosis? Should he be forbidden competitive games?*

A.—It may be presumed that this young man has rheumatic aortic incompetence, but it is by no means clear just how much the valve is damaged. If the leak is mild, no restriction of effort is necessary, and the prognosis is good. If the leak is moderate, however, strenuous competitive effort is best avoided, because, although at the present time he may be capable of 20-mile marathons without symptoms, such a great effort is likely to encourage cardiac enlargement and to shorten the symptom-free period. The prognosis of aortic incompetence of moderate grade is still fairly good if the heart is not subjected to long periods of strain. With so good an effort tolerance it is improbable that this young man has severe aortic incompetence.

During the symptom-free phase of aortic valve disease the severity of the damage cannot be assessed on effort tolerance, but attention should be paid to the character of the arterial pulse, to the blood pressure, to the size of the left ventricle clinically and radiologically, and to the electrocardiographic appearances.

Acetylcholine in Anxiety States

Q.—*Have drugs with an antagonistic action to adrenaline any place in the treatment of anxiety states, in particular the so-called adrenaline syndrome?*

A.—There is no drug which is solely an antagonist to adrenaline, and the only condition in which over-production of adrenaline is the main pathogenic feature is a specific form of suprarenal tumour. In nearly all anxiety states the disturbance is of the autonomic system as a whole, and, though sympathetic overaction may be dominant, some overaction of the parasympathetic system will be seen in individual symptoms.

Many physical methods of treatment in psychiatry probably owe their usefulness to their effect on the vegetative nervous system; electro-shock treatment, for instance, stimulates the sympathetic system, which is usually underactive in states of depression. Drugs which have a useful effect on sympathetic overactivity, and are also useful in the treatment of anxiety states, are barbiturates, insulin, and acetylcholine. Of these, acetylcholine is the nearest to being an antagonist to adrenaline, and after its administration the concentration of adrenaline in the blood tends to fall. Acetylcholine is now being used for the treatment of anxiety states in a number of centres in this country, and has been applied in a large series of patients by Lopez Ibor in Madrid. Professor Lopez Ibor recently read a paper on the subject to the Psychiatric Section of the Royal Society of Medicine. Experience in this country is not yet long enough for the indications for this treatment to be clear, but certainly individual patients are found who benefit very much.

Atheroma and Cerebral Accidents

Q.—*What is the relationship between atheroma and cerebral vascular accidents? How may atheroma be diagnosed clinically?*

A.—Atheroma of the cerebral arteries is an important predisposing cause in cerebral thrombosis and in the cerebral softening which may occur with narrowing of the lumen of cerebral arteries without actual thrombus formation; it is probably of less importance in the aetiology of cerebral haemorrhage. A rare cause of cerebral embolism is the lodgement in a cerebral artery of a fragment detached from an atheromatous plaque in the aorta. The clinical diagnosis of atheroma of the cerebral arteries is usually largely presumptive, but its presence is suggested by the occurrence of one or more thromboses in the absence of hypertension, syphilis, or other conditions which predispose to thrombosis, and by the radiological demonstration of dilatation and calcification in the aorta.

Cervical Spondylo-arthrosis

Q.—*What is cervical spondylo-arthrosis?*

A.—Cervical spondylo-arthrosis is more generally—though less accurately—known as osteoarthritis of the cervical spine. It begins as a degenerative lesion of an intervertebral disk (not necessarily associated with prolapse of the disk) and affects secondarily the posterior intervertebral (facet) joints. It may be confined to one segment or affect several. By the time significant symptoms arise *x*-ray changes are usually apparent. They consist, at first, in narrowing of one or more of the intervertebral disk spaces. Later there is lipping or spurring of the adjacent vertebral bodies and irregularity, narrowing, and osteophyte formation in the posterior intervertebral (facet) joints. These latter changes are seen well only in oblique projections. The cause of cervical spondylo-arthrosis, like that of osteoarthritis elsewhere in the spine, is degeneration of one or more of the intervertebral disks, either from "wear and tear" or from a specific injury; but the condition does not usually develop for many months or even years after the injury.

Clinically there are pain and stiffness of the neck, with sometimes a radiating pain down one upper limb from interference with a nerve root. There are periodic exacerbations of the pain—probably precipitated by some mechanical strain—with relatively mild persistent symptoms between the attacks. The pain responds in varying degree to physiotherapy, rest in a plaster collar, or sometimes manipulation. In most cases the disability persists intermittently for an indefinite period, but seldom becomes seriously incapacitating. In a few cases fusion of the affected part of the spine by operation is required for relief of severe symptoms.

Head Injury and Drunkenness

Q.—*In what ways may (a) very recent or (b) previous concussion modify the results of tests for drunkenness?*

A.—A previous answer in "Any Questions?" (June 10, 1950, p. 1381) deals partially with this problem. A "very recent" head injury can make the usual clinical tests for drunkenness largely useless, as confusion, difficulty in speech, incoordination of movements, and reflex changes can all result from a recent blunt head injury. The problem of a head injury some considerable time previously is more difficult, but it is well recognized that people who have had a head injury of any severity are often for a period more susceptible to the effects of alcohol than are normal people, and the signs of alcoholic intoxication may appear with a considerably smaller amount of alcohol than would produce them when the person is in his normal health.

Snags in Bacteriological Diagnosis of Tuberculosis

Q.—*Some years ago work was published describing errors in the diagnosis of pulmonary tuberculosis caused by artifacts which assumed the appearance of tubercle bacilli when sputum collected in rubber-capped bottles was stained and examined by ordinary methods. Has this work been confirmed, and, if so, what precautions are taken to avoid the error by present-day bacteriologists? Sputum is still collected in rubber-capped bottles.*

A.—The "artifacts" referred to are non-pathogenic acid-fast bacilli of the type occurring in soil and water. These have been found in large numbers on some batches of bark corks, and less often on rubber corks, particularly when the surface of the rubber is perished and cracked. The remedy is not to employ either of these materials for the closure of sputum containers: glass-stoppered bottles or cartons are to be preferred. Any unexpected finding of apparent tubercle bacilli should be confirmed by repeated examination and preferably by cultivation: the cultural characters of non-pathogenic *Mycobacteria* are entirely different from those of *Myco. tuberculosis.*

Tinnitus in the Elderly

Q.—*What treatment is advised for long-standing tinnitus in elderly patients?*

A.—Tinnitus is a common and in itself a harmless complaint. It may be caused by some disturbance of any part of the auditory apparatus, and in its more troublesome form is seen when the internal ear is at fault. As the years advance tinnitus is more frequently noted, and it is probable that the small continuous noises of later life are the result of the organ of hearing being insufficiently nourished, due to some vascular disturbance. That is why peripheral vasodilators such as nicotinic acid have had such a vogue in the treatment of tinnitus, though the results are often disappointing. In other cases a toxin may be responsible, and one that is occasionally encountered is the toxic effect of salicylates. Only a few people are sensitive to salicylates, but when they are it usually takes the form of tinnitus and possibly deafness. Many patients fear that tinnitus may mean some serious disturbance within the central nervous system, possibly one that may end in insanity. For this reason it is important to explain to patients that tinnitus is not harmful, and it does not imply organic disease within the central nervous system. It is only organized noises such as voices, bells, hymn tunes, etc., which rightly give rise to suspicion of a psychiatric background.

Firm reassurance, supplemented if necessary by a gentle sedative, is usually sufficient to comfort a patient suffering from tinnitus. Destructive operations on the ear rarely help, and should never be advised when tinnitus is the principal symptom. In the rare event of the tinnitus being unbearable prefrontal leucotomy may have to be considered.

Congenital Cataract

Q.—*What is the best treatment for congenital cataract in an infant of 6 months? When should it be carried out, and what is the prognosis? Both eyes are affected, the right considerably more than the left.*

A.—The question is not easy to answer, since the exact degree of the cataract is not stated. There are very many people with apparently normal vision who have congenital cataracts composed of dot-like opacities which are not sufficient in number or extent to inconvenience the patient at all. Between this and the complete congenital cataract which precludes any useful vision there are many stages. If the cataract in both eyes is of the latter type, the worst eye should be operated on, otherwise mental retardation will occur. If, however, the better eye will be capable of 6/18 or better vision, the operation should not be attempted, since 6/18 vision with accommodation is better than 6/6 without accommodation.

Brain Foods

Q.—*Is there any truth in the view that first-class protein, such as cheese, fish, and pig's fry, is specially indicated for "brain-workers"?*

A.—There is no evidence that these foods are specially required for the metabolism of the brain. It may be argued, however, that the mental worker requires about the same allowance of protein as the manual worker for the replacement of tissue wastage, but less carbohydrates and fat to produce energy. His diet should therefore contain a higher proportion of protein so as to bring the daily intake to about the same level in each case. The mental worker, moreover, may come to the table less hungry than the manual worker, and the liberal use of savoury foods, rich in animal protein, may help to stimulate his more fickle appetite.

Coal Tar and Pine Tar Soap

Q.—*Does the regular use of coal tar or pine tar soap predispose to light sensitization or epithelioma?*

A.—No.

Drunk in Charge

Q.—*Where can I find information how to examine a person suspected of being under the influence of alcohol while in charge of a car?*

A.—On p. 66 of the book "*Any Questions?*" The book can be obtained for 7s. 6d. (postage 6d.) from the Publishing Manager, B.M.A. House, Tavistock Square, W.C.1.

All communications with regard to editorial business should be addressed to THE EDITOR, BRITISH MEDICAL JOURNAL, B.M.A. HOUSE, TAVISTOCK SQUARE, LONDON, W.C.1. TELEPHONE: EUSTON 4499. TELEGRAMS: *Aitiology, Westcent, London.* ORIGINAL ARTICLES AND LETTERS forwarded for publication are understood to be offered to the *British Medical Journal* alone unless the contrary be stated.

Authors desiring REPRINTS should communicate with the Publishing Manager, B.M.A. House, Tavistock Square, W.C.1, on receipt of proofs. Authors overseas should indicate on MSS. if reprints are required, as proofs are not sent abroad.

ADVERTISEMENTS should be addressed to the Advertisement Manager, B.M.A. House, Tavistock Square, London, W.C.1 (hours 9 a.m. to 5 p.m.). TELEPHONE: EUSTON 4499. TELEGRAMS: *Britmedads, Westcent, London.*

MEMBERS' SUBSCRIPTIONS should be sent to the SECRETARY of the Association. TELEPHONE: EUSTON 4499. TELEGRAMS: *Medisecra, Westcent, London.*

B.M.A. SCOTTISH OFFICE: 7, Drumsheugh Gardens, Edinburgh.